HEALTH ASPECTS OF PESTICIDES

abstract bulletin

September 1972 - Vol. 5, No. 9

Abstracts 72-1811-2037

HAPAB 05(9) 421-473 (1972)

Quarterly Index Issue

ENVIRONMENTAL PROTECTION AGENCY

ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDES PROGRAMS

DIVISION OF PESTICIDE COMMUNITY STUDIES
S. W. Simmons, *Director*
4770 Buford Highway, Bldg. 29
Chamblee, Georgia 30341

HEALTH ASPECTS OF PESTICIDES ABSTRACT BULLETIN is a monthly publication of the Division of Pesticide Community Studies of the Environmental Protection Agency.

The purpose of this publication is to foster current awareness of the major worldwide literature pertaining to the effects of pesticides on humans. The publication represents a monthly review of more than 1150 domestic and foreign journals. Subjects and authors of abstracted articles are cross-referenced in an annual index issue. Reprint requests should be directed to the author of the complete article.

Publication of an abstract, annotation, or citation does not imply endorsement by the Environmental Protection Agency of the original article or of commercial materials, equipment, or processes mentioned therein.

CONTENTS

Vol. 5 September 1972 No. 9

Page

General 421

Monitoring and Residues 426

Epidemiology, Prevention and Treatment 437

Toxicology and Pharmacology 443

Analysis 466

Quarterly Index (July-September)
- Preface I-1
- Subject Index
  - I. Compounds I-2
  - II. Concepts I-21
- Author Index
  - I. Personal I-44
  - II. Corporate I-56

72-1811. Nicholson, H. P. (Southeast Water Lab., FWPCA, USDI, Athens, GA). **The pesticide burden in water and its significance.** *In: Agricultural Practices and Water Quality, T. L. Willrich and G. E. Smith, ed., Iowa State Univ. Press, Ames, Iowa, 1970, pp. 183-193.* (32 references)

Persistent pesticides present a greater threat to water quality than readily degraded compounds. Dieldrin, endrin, DDT, and DDE have a widespread distribution in U.S. waterways at concentrations < 1 ppb. Generally, bottom sediments display much higher pesticide concentrations than water, suggesting that standards for overall quality of a stream should deal with the total pesticide burden rather than concentrations in water. Agricultural runoff is the chief source of low-level contamination and varies with the nature of the pesticide, soil type, land use, topography, and climate. Industries which manufacture, formulate, or use pesticides or reclaim used pesticide containers are probably the second most significant source of pesticides in water. Accidents, carelessness, airborne dust, and direct application are all possible sources of contamination. Minimization of use of persistent chemicals, treatment of industrial effluents, use of biological controls and farm planning based on the Universal Soil Loss Equation would reduce the level of pesticide pollution.

72-1812. Peters, D. C. (Dept. of Zool. and Entomol., Iowa State U., Ames, IA). **Pesticides and pest management for maximum production and minimum pollution.** *In: Agricultural Practices and Water Quality, T. L. Willrich and G. E. Smith, ed., Iowa State Univ. Press, Ames, Iowa, 1970, pp. 209-223.* (45 references)

Controls applied for the management of pests are quite varied, and agencies concerned with them are attempting to insure minimal environmental effects. More imaginative field research and, consequently, more money are needed. A number of examples of alternate control methods and practical uses illustrate present capabilities and the problems encountered. The introduction and encouragement of parasites and predators of pests and timely application of synthetic compounds are very effective means of control. Herbicides are used on a higher percentage (>50%) of the corn and grain acreage in the United States than insecticides. Although use of pesticides with livestock represents only 5% of the total farm use, misuse and contamination are problems involved. The use of insecticides on fruit and cotton is generally high, and in the latter case, they are not subject to large dilution on entering the environment. Only about 5% of the forest lands are sprayed, but wildlife in the areas may be significantly affected. Home use is a source of water contamination since leftover pesticides can easily end up in sewage systems. Legislative action could reduce the overall pesticide burden by encouraging planting of crops in areas where specific pests have not occurred and doing away with the maximum yield per acre incentives. Federal programs aimed at reducing costs have also reduced target precision and effectiveness.

72-1813. Campbell, R.; Whitley, J. R. (U. of Missouri, Columbia, MO). **Effects of agricultural pollutants on recreational uses of surface waters.** *In: Agricultural Practices and Water Quality, T. L. Willrich and G. E. Smith, ed., Iowa State Univ. Press, Ames, Iowa, 1970, pp. 331-343.* (43 references)

Agricultural pollutants including pesticides have a profound impact on the aquatic community, and thus on the recreational potential of water. Biological magnification of pesticides and the related problem of large residues resulting from development of resistance in certain species are of concern from the viewpoint of both the detrimental effects on aquatic life and the hazardous levels of chemicals in man's food. The persistence of organochlorine pesticides intensifies accumulation problems. Water quality standards recently adopted by the states have established minimal limits for the addition of agricultural pollutants to State waters. The Report of the National Technical Advisory Committee to the Secretary of the Interior is a standard reference for water quality criteria which will protect water for recreational and other uses.

72-1814. Hines, N. W. (U. of Iowa, Iowa City, IA). **Legal aspects.** *In: Agricultural Practices and Water Quality, T. L. Willrich and G. E. Smith, ed., Iowa State Univ. Press, Ames, Iowa, 1970, pp. 365-376.*

The legal requirements in the area of water pollution from agricultural sources are reviewed, and ways in which the law could affect future agricultural pollution policy are suggested. Few cases of agricultural pollution have been handled under the "nuisance theory." Since such cases are generally hard to win, reliance on public pollution control agencies is increasing. Municipal and industrial pollutors seem to be the present targets of Federal and State agencies. Point sources such as feedlots and heavy applications of pesticides have been of some concern, but the effects of soil erosion, animal and crop wastes, and chemical fertilizers have received much less attention. Pesticides, at the time of writing, were Federally controlled only through tolerances in foods set by the FDA and through the USDA. One of the prime problems that lawyers face in environmental defense litigation is that of finding scientific data on the relative toxicity of different chemicals, a problem which public hearings would improve. In the future, more attention will probably be given to requiring better land-use practices and substitution of less toxic chemicals or biologic techniques for hard pesticides. No legal constraints exist for the enactment and enforcement of such requirements.

72-1815. Legator, M. S. (George Washington U., Washington, DC). **Chemical mutagens.** *Annu. Rev. Med. 23: 413-428; 1972.* (32 references)

The majority of mutagenic agents could be detected by concurrent application of three procedures developed in recent years, *i.e.* the host-mediated assay,

the dominant lethal test, and *in vivo* cytogenetic studies. These comparatively simple, economical procedures are as relevant to man as other currently used toxicological methods and should be improved upon and utilized in evaluation of all drugs, pesticides, and other commercial chemicals. These chemicals probably play a more important role in mutations than do physical agents, viruses, or so-called spontaneous mutations. At present there is no meaningful monitoring of populations for induced mutations.

72-1816. Bergmann, E. D. (Dept. of Organic Chem., Hebrew U., Jerusalem, Israel). **Die Zukunft der Insektizide—ein Umweltproblem. [The future of insecticides—an environmental problem.]** *Chem. Unserer Zeit 5(5): 142-146; 1971.* (3 references) (German)

The insecticides represent an illustrative example of the environmental problems facing man as a result of continuing technological development. This development was initiated as a consequence of man's desire to improve his living conditions, but has proceeded to a point where technology may negate its own gains. Chemical insecticides are indispensable at present for disease control and maintenance of food production, but the search for alternate control means must be continued. Alternate controls are necessitated by the fact that accidental contamination of the environment by excessive or inappropriate pesticide use is unavoidable, and by the development of pesticide-resistant insect species. Progress in the use of sterilization techniques, sexual lures, and insect hormones promises that it will be possible to reduce, but not eliminate, the use of chemical insecticides. More research is needed to guide the battles against pests and pollution, and technology must solve the problems it has created.

72-1817. Wright, R. M.; Bowen, C. E. (Geology Dept., Stanford U., Palo Alto, CA). **Water pollution: an environmental health hazard.** *J. Sci. Res. Counc. Jam. 2(2): 66-89; 1971.* (31 references)

A review of some sources of water pollution and an outline of approaches to solution of the problem are presented. Standards for domestic water supplies may soon include pesticides and herbicides. At present, limits have been established for arsenic, lead, and other hazardous elements. Pesticides and herbicides reach water supplies as a result of application for pest control in the water or from runoff from cultivated areas, careless spraying practices, and industrial waste. Toxicity of the organochlorines, the organophosphates, and herbicides has been documented. More research is needed to determine what levels in water can be hazardous in the long run to man and the ecosystem. Persistent organochlorines should be phased out and replaced by rapidly degrading compounds, and pesticidal uses of mercury should be eliminated where safer substances can be used.

72-1818. Bour, H.; Kreybig, T. von (Abt. fuer Moraltheologie, Katholische Theologische Seminar der Universitaet, 74 Tuebingen, Germany). **Toxikologie und Ethik. [Toxicology and ethics.]** *Med. Klin. (Munich) 65(15): 751-757; 1970.* (32 references) (German)

The ethical ramifications of man's attempts to improve his living conditions, prevent disease, and avoid the pains and stresses of life by technological means are discussed. At first many technological measures, such as the use of insecticides and drugs, were taken without full knowledge of the consequences, and a number of untoward incidents resulted. Among these incidents were the upsetting of the balance of nature due to nonselective destruction of insects and the harmful effects observed on wildlife after the use of insecticides. Direct threats to man may exist in the induction of microsomal drug-metabolizing enzymes and the suggested production of cancer by DDT. Classical ethics demands that in the case of an action with dual (positive and negative) effects, moral priorities should be employed in making the ultimate decision. The priorities relating to insecticide use can be stated: priority of human health in the long run over momentary well-being or enjoyment; priority of effectiveness of the agent over its toxicity; priority of prevention over the impulse to experiment on man; and priority of humanitarian interests over purely economic and personal ones.

72-1819. Obst, A. (Bayerische Landesanstalt fuer Bodenkultur und Pflanzenbau, Germany). **Die Bekaempfung parasitaerer Erkrankungen im Ackerbau aus der Sicht des integrierten Pflanzenschutzes. [The control of parasitic diseases in agriculture from the viewpoint of integrated plant protection.]** *Nachrichtenbl. Deut. Pflanzenschutzdienstes 24(5): 65-70; 1972.* (42 references) (German)

In the control of fungal diseases of plants, excessive or inappropriate use of fungicides is not only disadvantageous from the viewpoints of economics and of environmental pollution, but may even increase the susceptibility of the plants to such diseases. This may be brought about either by an impairment of natural control mechanisms such as competitive inhibition or by the production of resistant fungi. The broad-spectrum fungicides, *e.g.*, the mercurials, are more likely to cause the former effect, while the newer heterocyclic hydrocarbon fungicides, which attack only one point in the metabolic cycle, have a greater tendency for the latter. An integrated control system will reduce the amounts of fungicide required, as well as increase the effectiveness of the treatments applied. The concept of soil hygiene, defined as the maintenance of the soil in a condition most conducive to the development of healthy crop plants, includes the utilization of crop rotation and optimal nutrient supply. Plant breeding has proved particularly effective against some fungal diseases. The schedule of grain planting and fungicide application has a great effect on fungal disease incidence.

72-1820. Beitz, H.; Angermann, R.; Becker, H. G. (Institut fuer Pflanzenschutzforschung Kleinmachnow der Deutschen Akademie der Landwirtschaftswissenschaften zu Berlin, DDR). **Pflanzenschutz und Umweltschutz unter den Bedingungen der Intensivierung der Pflanzenproduktion. [Plant protection and environmental protection under the conditions of intensification of plant production.]** *Nachrichtenbl. Pflanzenschutzdienst DDR 26(5): 87-92; 1972.* (8 references) (German)

The desire to improve food supplies in East Germany, as in the rest of the world, is expressed in an intensification of agricultural production. This implies not only more extensive but also more efficient utilization of agricultural chemicals and necessitates great care in the prevention of environmental pollution. Increased efficiency is expected to result from the formation and training of special agrochemical brigades and orderly direction of agrochemical applications by government agencies. Environmental protection is viewed in East Germany as a natural outgrowth of the socialist cooperative planning system. State research institutions are charged with the study of agricultural chemicals and their effects and with resolving conflicts such as the need for prolonged efficacy versus the avoidance of undesirable residues of pesticides. The legal foundations for environmental protection efforts in East Germany were laid by such general laws as the Constitution of 1968 and the Agriculture Regulations of 1970 and specific laws such as the plant protection law of 1953, the occupational health and fire prevention law of 1968, and regulations governing the use of aircraft in agriculture (1970-71), protection of drinking water supplies (1970), and protection of bees (1951).

72-1821. Lembcke, G.; Haselein, I. (Pflanzenschutzamt und Institut fuer Veterinaerwesen beim Rat fuer Landwirtschaftliche Produktion und Naehrungsgueterwirtschaft des Bezirkes Schwerin, DDR). **Pflanzenschutzmassnahmen im Winterraps unter Besonderer Beruecksichtigung des Umweltschutzes. [Plant-protection measures in winter rape with particular reference to environmental protection.]** *Nachrichtenbl. Pflanzenschutzdienst DDR 26(5): 97-100; 1972.* (16 references) (German)

The intensive production of rapeseed oil, which is of great economic importance in northern East Germany, requires heavy pesticide application. The major rape pests are controlled by aerial application of DDT, lindane, and toxaphene, and a number of problems have arisen as a result of spray drift from these applications. Agrochemical application planning, including careful selection of dates for insecticide application, consideration of weather conditions, and selection of insecticides according to the contamination danger in a given area, has reduced the danger of poisoning bees, fish, and animals. To prevent food and feed contamination, further measures are recommended: concentration of rape cultivation areas, eliminating interspersal with dairy farms, etc.; driving cattle away from areas to be treated; and if possible, mowing or harvesting of areas adjacent to rape fields before spraying. If drift into adjacent fields does occur, an interval of 42 days must be allowed before cattle are returned to these areas, unless earlier residue determinations by the regional veterinary institute indicate that the field is safe for grazing. Elevated DDT levels in the Warnow River were recorded on the day after DDT treatment of rape fields in the Schwerin district. Therefore, the use of the low-toxicity preparation Fekama AT (active ingredient: butonate) is now required where high runoff is expected.

72-1822. Maddox, J. (Author address not given). **Pollution and worldwide catastrophe.** *Nature (London) 236(5348): 433-436; 1972.* (13 references)

A review of pesticide pollution by organochlorines indicates that the persistent pesticides are more a means for survival of the human race, with some unpleasant or unexpected side effects which need to be investigated, than a threat to its survival. Although DDT accumulates in human fat, levels appear to reach an equilibrium and are not sufficiently high to warrant concern that the toxicological effects observed in animals will appear. Some of the original assumptions regarding carcinogenic properties have been undermined by more recent data. Statistics on many phases of DDT pollution leave much to be desired.

72-1823. Anonymous. **DDT condemned.** *Nature (London) 237(5356): 422-423; 1972.*

The recent decision of William Ruckelshaus of the Environmental Protection Agency to eliminate most domestic uses of DDT by December 31, 1972, was followed by two court actions. One action hopes to put the ban into effect immediately on the grounds that growers are already accustomed to using methyl parathion, the likely substitute for DDT, and the time allowed for this adjustment is unnecessary. The other action is trying to have the ban repealed. Years of debate have not yet resolved the question of the risks of using DDT. The courts have in the past decided in favor of the EPA, and although industry has taken its case to the cotton belt, it is not expected to succeed in its efforts to change the decision.

72-1824. Iliff, N. (Author address not given). **Organic chemicals in the environment.** *New Sci. 53(781): 263-265; 1972.*

Approximately 20 million tons of manufactured organic chemicals may be put into the environment annually, including about 1 million tons of organic pesticides. Much more research is needed on distribution and toxicity of these compounds, particularly in the field of persistent chemicals. Monitoring systems of much

greater sophistication and international operation will be important for future studies.

72-1825. Foster, G. G.; Whitten, M. J.; Prout, T.; Gill, R. (Div. of Entomology, Commonwealth Scientific and Industrial Research Organization, Canberra, Australia). **Chromosome rearrangements for the control of insect pests.** *Science 176(4037): 875-880; 1972.* (26 references)

Current theory on the control of insect pests by genetic manipulation is reviewed. An outline is given of the synthesis and behavior of a particular type of chromosome rearrangement, called a compound chromosome, for altering the genetic composition of natural populations of a particular pest. In effect, the process aims to displace field populations of the pest through the release of a new form where premating isolation behavior is absent but postmating isolation, through genetic death of the hybrid, is complete. This new form would either carry a conditional lethal genotype or remove the noxious features of the pest, such as vector capacity. Displacement of the old form follows from the instability generated by the hybrid lethality and could take as little as five generations for completion. It is claimed that pest control by genetic displacement parallels a common evolutionary process. The list of pests susceptible to this form of control is limited, but includes several important pest species such as mosquitoes and other dipterans. (Author abstract by permission. Copyright 1972 by the American Association for the Advancement of Science)

72-1826. Gillette, R. (Author address not given). **DDT: its days are numbered, except perhaps in pepper fields.** *Science 176(4041): 1313-1314; 1972.*

The turning point in the history of the use of DDT in the United States was the shift of Federal pesticide authority from the Department of Agriculture to the Environmental Protection Agency. Since that time, with the help of pressure from environmentalists and the Environmental Defense Fund, virtually all domestic uses of DDT except those in public health will have been banned by December 31, 1972. Unless growers of the three minor crops excluded from the ban, including peppers from the Delmarva peninsula, can present new evidence to support the use of DDT within one month, a total ban will go into effect. The two court appeals in progress are not expected to reverse the ban.

72-1827. Entomological Society of Canada. (Author address not given). **Pesticides and the environment.** *Bull. Entomol. Soc. Can. 3(1); 1-16; 1971.*

To provide the Canadian public with objective information concerning the problem of environmental pollution by pesticides, the Entomological Society of Canada issued a statement proposing short- and long-term solutions. The pesticide problem, like other forms of pollution, is the result of the excessive demands placed by man on a finite environment. The long-term solution requires the reduction of man's numbers and consumption of resources to a level where adequate health and comfort can be maintained without deterioration of the environment. The short-term solutions mainly involve regulating the way in which pesticides are applied and reducing the amount of pesticides used. Improvements are needed in the licensing of pesticide applicators and in the mechanism of pesticide registration, as well as in the systematic monitoring of pesticide effects. The quantities of pesticides used can be reduced by promoting the concept of using the minimum amount necessary for a given purpose, encouraging the development of alternate control methods, and informing the public about the causes and prevention of pollution.

72-1828. Clausen, J. (Neurokemisk Inst., Universitetets Hyg. Inst., Radmansgade 58, DK-2200, Copenhagen, Denmark). **Foedemiddelforurening. [Food contamination.]** *Nord. Med. 86(50): 1501-1507; 1971.* (32 references) (Danish)

The mechanism and physiological consequences of the contamination of food due to biocides, radioactivity, chemicals, and microorganisms are reviewed. A mathematical description of the process of biocide accumulation in the ecological system is given, with special regard to lipid-soluble pesticides with limited metabolism. The effects of pesticides are determined by concentration, solubility, and stability. The DDT concentration in the biomass is directly proportional to age and retention and inversely proportional to the total mass. Lipid-soluble organochlorine pesticides concentrate by a factor of about 2000 while passing from one stage of the food chain to another. The DDT concentrations measured in dead cancer patients were 2-3 times higher than normal. Effects on animals were observed at 20-60 ppb concentrations. Plant photosynthesis was inhibited by DDT at a concentration of 2 ppb. Certain bacteria were found to decompose DDT. Organic mercury compounds have concentration factors of about 8000 and 4000 in invertebrates and fish, respectively. The maximum acceptable daily mercury intake is 0.028 mg. While the mercury residue contents in meat products and eggs usually lie far below the acceptable limit, high contents in fish are often found.

72-1829. Hontani, I. (Faculty of General Culture, Tokyo U. of Agr. and Technol., Tokyo, Japan). **[Military defoliation in Vietnam and the scientist's responsibility.]** *Seibutsu Kagaku (Biol. Sci.) 23(3): 155-160; 1972.* (10 references) (Japanese)

Although military defoliation in Vietnam using 2,4,5-T has been stopped, other agents, particularly picloram, continue to be used. According to a report by

the Library of Congress, a total area of 1,668,584 ha received defoliant treatment between 1962 and March 1969; this included 189,766 ha of crop area. Such applications are considered objectionable by ecologists and other scientists, in Japan and elsewhere, from two points of view: first, the possibility of a direct hazard to man and his progeny, especially in view of the teratogenic effects of 2,4,5-T. The second is environmental destruction, whose long-term effects are not yet foreseeable and whose short-term effects such as crop destruction have already caused much human suffering.

72-1830. Takahashi, I. (First Dept. of Sanit. Eng., Kanagawa Prefectural Hyg. Inst., Japan). **[Pollution of soils and means for controlling it. III. Pesticide pollution.]** *Mizu Shori Gijutsu (Water Purif. Liquid Wastes Treat. (Osaka)) 13(6): 1-8; 1972.* (29 references) (Japanese)

Recent developments in the field of environmental pesticide pollution in Japan are reviewed, with particular reference to those pesticides which persist for long periods in soil and water. The law governing pesticide registration and use in Japan was amended in 1971; it now includes standards for safe pesticide use and residue tolerances for eight major pesticides in 16 major crops. General principles regarding the action and properties of the major pesticide groups and their fate in crops and soils are presented. Means of reducing environmental pollution by persistent pesticides are also outlined; these include the development of rapidly degrading pesticides, utilization of natural enemies of pests, and breeding of pest-resistant crops.

72-1831. Soller, B. (Author address not given). **Ville fraemja fisket. Roen om miljoegifter hade motsatt effekt. [Pesticide residues in sea fish and birds.]** *Laekartidningen 68(14): 1593-1600; 1971.* (Swedish)

Investigations of DDT, polychlorinated biphenyl (PCB), and mercury residues in Baltic fish and birds are described. The DDT and PCB residues exceeded the FAO/WHO-approved standards in several cases. Baltic dogfish had DDT contents 10 times as high as their counterparts from the shores of Canada, Great Britain, or the Netherlands. Herrings and anchovies from the Southern Baltic contained 2.3 mg and salmon, 3.4 mg of DDT per kg body weight. Cod livers contained 20-30 mg of DDT per kg. DDT residues in sea eagle brain were 100 mg/kg of fresh tissue. The mercury content in bird feathers was found to be a suitable method for investigating the variations in mercury concentrations in the environment. Feathers grown by birds while in Africa contained much less mercury than those developed in contaminated areas in the Northern hemisphere. There is a definite relationship between human blood and hair mercury level and mercury contents in fish. A concentration of 0.2 mg/kg body weight in mackerel can be regarded as a natural mercury level. Mercury is readily bound by acid media.

72-1832. Dencker, I.; Persson, K.; von Rosen, G. (Author address not given). **Resthalter av DDT och dess metaboliter i normalkost. [DDT and DDT metabolite residues in normal diet.]** *Laekartidningen 68(36): 4034-4036; 1971.* (7 references) (Swedish)

Residues of DDT, DDT metabolites, BHC, aldrin, dieldrin, and lindane in normal diet samples were determinated by gas-liquid and thin-layer chromatography. The residue contents in all 55 samples were near the detectability limit with a few exceptions. The DDT contents, ranging from 0.01 to 0.09 ppm, exceeded a value of 0.03 ppm in five cases and were lower than 0.02 ppm in nine cases. The *p,p'*-DDE and *p,p'*-DDD (TDE) residue contents ranged from 0.003 to 0.075 ppm and from 0.005 to 0.035 ppm, while the *p,p'*-DDT contents were in a range of 0.001-0.06 ppm. While both aldrin and dieldrin were detectable in only one sample, lindane residues exceeded 0.010 ppm in two cases. The latter and other BHC isomers were identified, though in very small quantities, in almost every sample. The daily DDT intakes ranged from 0.10 to 0.22 mg, compared with the acceptable daily intake of 0.70 mg specified by the World Health Organization.

72-1833. Turk, A.; Haring, R. C.; Okey, R. W. (City Coll., City U. of New York, New York, NY 10031). **Odor control technology.** *Environ. Sci. Technol. 6(7): 602-607; 1972.* (5 references)

Odor control in pesticide manufacture, as in other industries, requires reducing the residues of pesticides, their precursors, byproducts, and incidental materials in air to levels below those at which they are organoleptically detectable. In the manufacture of DDT, vapors of alcohol, chlorine, chloral, chlorobenzene, sulfur dioxide, and hydrochloric acid may be emitted from various parts of the operation. It is generally possible to remove 95% of the acidic gases from the air with a water scrubber. During the washing and steaming of the finished DDT, scrubbing with water or caustic soda solution will provide adequate control. Gases released during the manufacture of 2,4-D include vapors of phenol, chlorine, chloroacetic acid, dichlorophenol, and hydrochloric acid. An alkaline scrubber (to remove acidic gases, including phenols) is probably best in this case. Other methods used in the manufacture of some pesticides include air oxidation, dispersion, adsorption, and masking.

72-1834. Nakanishi, M.; Kuriyama, T.; Kudo, A. (Res. Labs., Yoshitomi Pharmaceutical Ind., Ltd., Yoshitomi, Fukuoka, Japan). **[Stability of a new pyrethroid: Kikuthrin.]** *Bochu-Kagaku (Sci. Pest. Contr.) 35(3): 96-102; 1970.* (10 references) (Japanese)

The decomposition of proparthrin, a synthetic pyrethroid, as the pure substance before formulation into mosquito coils, is mainly due to oxidative degradation. The decomposition rate is proportional to the specific surface area. Proparthrin can be stabilized by the addition of an antioxidant, such as butylhydroxytoluene (BHT), or by storage under nitrogen. Kikuthrin is the trade name given to the formulation containing proparthrin plus 1% BHT, which is sufficiently stable for storage under practical use conditions. Proparthrin is readily hydrolyzed in alcoholic alkaline solution due to the ester group contained in its molecule. However, the rate of hydrolysis in aqueous suspension is relatively low. The Kikuthrin formulation did decompose under drastic conditions, such as exposure to direct sunlight in August. However, under normal conditions, Kikuthrin mosquito coils are anticipated to remain stable for about 1 yr. Proparthrin was the only volatile product found in the smoke from Kikuthrin mosquito coils; no volatile decomposition products were found.

72-1835. Kamada, T. (School of Hyg., Fac. of Med., Hiroshima U., Hiroshima-shi, Japan). **[Hygienic studies on pesticide residues. Part 2. Study on the source of BHC contamination of milk.]** *Nippon Eiseigaku Zasshi (Jap. J. Hyg) 26(4): 365-370; 1971.* (5 references) (Japanese)

Residues of organochlorine pesticides, particularly BHC isomers and DDT, in cows' milk have posed a serious public health problem in Japan. To determine the source of this contamination, BHC isomer contents in milk, fodders and feeds, soils, and tissues from cows in Hiroshima Prefecture were examined. Rice straw was found to be the main source of the contamination, accounting for 77% of the total BHC and 95% of the β-isomer found in the milk. High contents of BHC

(0.26-0.36 ppm total BHC, 0.22-0.56 ppm β-isomer) were found in the milk of cows from districts where large amounts of BHC were used in rice growing. The BHC on the rice straw was accounted for by pesticide which adhered to the stem at the time of application, not by uptake from the soil. BHC was recently banned in Japan, and soil levels in formerly polluted regions have already declined almost to equal the previously nonpolluted areas. Therefore, once the directly contaminated rice straw is eliminated, the BHC levels in the milk will be expected to decline. The β-isomer appears to be excreted rapidly in the milk after ingestion, so that adipose tissue BHC levels will probably reach a minimal value soon.

72-1836. Kojima, K. (Sect. of Food Chem., Dept. of Environ. Hyg., Ministry of Public Welfare, Chiyoda-ku, Tokyo, Japan). **[Epidemiology of organochlorine pesticide pollution.]** *Baioteku (Biotech) 3(7): 589-592; 1972.* (6 references) (Japanese)

Of the various organochlorine pesticides used in Japan, BHC (technical grade, containing a mixture of isomers) was most widely used and was responsible for the greatest environmental pollution. A correlation was found between the quantities of BHC applied to rice paddies in certain areas (e.g., in Nagasaki Prefecture, 10-90 kg/ha between 1964 and 1970) and the total BHC content of cows' milk (reaching a maximum of 1.50 ppm in this same area). The main route of entry for this pesticide into the milk was found to be contaminated rice straw fed to the cows. Due to the increasing restrictions placed on BHC use and the total ban imposed in November 1971, the content of the most persistent isomer, the β-isomer, in milk is decreasing. Milk in Nagasaki prefecture contained a maximum of 2.028 ppm BHC in February 1970 and a maximum of 0.173 ppm in December 1971. Residues in beef are also declining. The total BHC content of fruits and vegetables in Tokyo was already found to be below 0.01 ppm in June 1970. In 1971, total diet studies revealed that the average Japanese ingests 1.75-2.89 μg/kg/day of total organochlorine pesticides, including 0.63-1.34 μg/kg/day total BHC and 0.22-0.46 μg/kg/day total DDT. This intake is comparable to the total of 1.29-3.19 μg/kg/day reported for Kansas in 1968, 0.24-1.14 μg/kg/day reported for Baltimore in 1968, and 0.43-4.25 μg/kg/day in England.

72-1837. Minagawa, K.; Yamamoto, N.; Yoneya, T.; Wakai, S. (Niigata Prefectural Inst. of Hyg., Niigata-shi, Japan). **[Organochlorine pesticide residues in standard diets.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Jap.) 13(3): 245-246; 1972.* (Japanese)

Three types of standard diets were prepared: one representing the diet on a farm in the mountain region of Japan, one from a farm on the plains, and one representing that of an urban family. Each consisted of cereals, meats, leafy and root vegetables, fruits, oils and fats, milk and milk products, eggs, dried beans and peas, and water. Samples representing these diets were collected in May, August, and November 1971 and February 1972. They were prepared for table, and the organochlorine pesticide contents determined by the FDA methods. The average total BHC intake was 26.1 μg/person/day on the plains farm (PF), 19.0 in the urban area (UA), and 18.4 on the mountain farm (MF). The total DDT intake was 11.4 μg/person/day (PF), 9.1 (UA), and 7.6 (MF). The dieldrin + aldrin intake was 6.0 μg/person/day (UA), 2.2 (MF), and 1.5 (PF). Seasonal variations in residue content were noted; for example, the BHC residues in meats were higher in autumn and winter, and DDT and dieldrin residues in meats were higher in winter. Meats accounted for the majority of the total residues in the diet: 32.0% of the BHC, 66.6% of the DDT, and 43.9% of the dieldrin. The calculated daily intake values were below the WHO/FAO recommended levels, except possibly for dieldrin.

72-1838. Kevorkian, J.; Cento, D. P.; Hyland, J. R.; Bagozzi, W. M.; van Hollebeke, E. (Saratoga Gen Hosp., Detroit, MI 48205). **Mercury content of human tissues during the twentieth century.** *Amer. J. Pub. Health 62(4): 504-513; 1972.* (16 references)

To provide a baseline for assessment of environmental pollution by mercury, organ specimens from 59 cases extending over 60 yr and representing both sexes and all ages were analyzed for mercury content. Methodological difficulties were anticipated, so samples were analyzed independently by three laboratories. An extremely wide range of variation in analytical results was found. Nevertheless, the results seem to indicate two broad and unexpected trends. The first is an apparent decline in the levels of mercury in human organs during the first three decades of the 20th century. The levels of mercury in brain, lung, kidney, and pancreas generally fell in the 30-ppm range in the earliest specimens studied, but dropped to the 5-10 ppm range in 1940's and 1950's. Levels in liver, spleen, adrenals, testes, uterus, and thyroid were lower initially (15-20 ppm), but showed a similar decline. The second finding was that the occurrence of mercury appeared to follow a biphasic curve pattern through progressive age decades, with peaks in early childhood and late middle age, which may be accounted for by differences in growth rates. The apparent decline in contamination of human organs by mercury may be related to the decline in coal burning, which shows a similar chronological pattern. High methylmercury levels in brain were not found.

72-1839. Reinert, R. E.; Stewart, D.; Seagran, H. L. (U.S. Fish and Wildlife Serv., Great Lakes Fishery Lab., Ann Arbor, MI 48107). **Effects of dressing and cooking on DDT concentrations in certain fish from Lake Michigan.** *J. Fish. Res. Bd. Can. 29(5): 525-529; 1972.* (3 references)

DDT residues were determined in yellow perch (*Perca flavescens*), bloater (*Coregonus hoyi*), lake trout (*Salvelinus namaycush*), and coho salmon (*Oncorhynchus kisutch*) captured in Lake Michigan. Samples were taken from raw fish (whole, eviscerated, filleted, or steaked) and from fish cooked by smoking, frying, baking, or broiling. Residues in all fish were highest in parts of the body with the highest oil content. Dressing reduced the DDT residues and oil content of yellow perch by more than 90%, but produced little change in the residues in the other three species. DDT residues in bloaters were not greatly affected by smoking, but were reduced 64-72% (from 8-10 ppm to 2-4 ppm) by other cooking methods. The concentration of DDT residues in fillets of yellow perch changed from 0.3 ppm to 0.4 or 0.5 ppm after baking, frying, or broiling. Initial DDT residues in fillets or steaks averaged: yellow perch, 0.3 ppm; bloater (medium), 10.0 ppm; coho salmon, 14.9 ppm; lake trout, 17.4 ppm.

72-1840. Anonymous. **Organophosphorus pesticides: residues and metabolism.** *Food Cosmet. Toxicol. 10(2): 243-246; 1972.* (13 references)

Recent findings regarding residues and metabolism of organophosphate pesticides are reviewed. Malathion is the organophosphate most frequently encountered in food; in one study, malathion residues of 1.6-7.4 ppm were found in Australian wheat, 0-3.8 ppm in wheat from eight other countries, and 0-2.0 ppm in corn samples. The grain dust was found to be particularly high in malathion, but caused no problems after processing. In total-diet studies in England and Wales, 20 of 462 food samples contained residues of demeton-S, disulfoton, fenchlorphos, fenitrothion, malathion, and parathion at 0.01-0.13 ppm. Malathion occurred most frequently, but at levels far below the WHO-recommended maximum acceptable daily intake. Residues of organophosphate insecticides found in foods in the United States were generally slightly higher than those found in the United Kingdom. Recent studies on organophosphate metabolism have emphasized the importance of the metabolic pathway followed for the formation or more or less toxic derivatives. Some findings have indicated the importance of reduced glutathione-dependent enzyme systems as primary or secondary detoxification routes for various organophosphates. Thus, in addition to the ability of reduced glutathione to conjugate with foreign compounds, it is important as an acceptor of alkyl groups released during the *O*-dealkylation of various compounds.

72-1841. Moergeli, B. (Winterthur, Switzerland). **Entfernung von Pestiziden aus Trinkwasser. [Removal of pesticides from drinking water.]** *Gas Wasser Abwasser 52(5): 142-146; 1972.* (13 references) (German)

Lindane-contaminated water specimens were prepared by adding a stock solution of lindane to water taken by a waterworks installation from Lake Constance in Switzerland. The addition of ozone at the level generally used for disinfection of lake water (0.4-3 mg/l, contact time 10 min) resulted in no reduction in lindane concentration. Sand filters, 80 cm thick, were also ineffective in removing lindane from water. Lindane was adsorbed to some extent at the beginning of the flow period, but retention decreased rapidly, and lindane continued to leach from the filter after the flow of contaminated water was changed to one of fresh water. Experiments with three types of activated charcoal showed that they possessed varying efficiencies in removing lindane from flowing water, but in general, a carbon layer 70 cm thick should be present to adsorb the majority of the lindane contained in lake water throughout an acceptable filter lifetime, reducing an initial lindane level of 50 ppb to about 1 ppb.

72-1842. Schuphan, I.; Sajko, B.; Ballschmiter, K. (Biologische Bundesanstalt, Institut fuer Pflanzenschutzmittelforschung, 1 Berlin 33 Dahlem, Germany). **Zum chemischen und photochemischen Abbau der Cyclodien-Insektizide Aldrin, Dieldrin, Endosulfan und weiterer Hexachlorbicyclo-[2.2.1]-hepten-Derivate. [Chemical and photochemical degradation of the cyclodiene insecticides aldrin, dieldrin, endosulfan and other hexachloronorbornene derivatives.]** *Z. Naturforsch. 27B(2): 147-156; 1972.* (23 references) (German)

The hexachloronorbornene moiety of the cyclodiene pesticides is highly resistant to degradation, but several routes to specific dechlorination of these compounds are known. Under conditions prevailing in nature, dechlorination at the bridge carbon with its geminal chlorine atoms seems most likely, followed by nucleophilic attack by a hydroxyl group or reductive dechlorination, then by oxidation. The liberation of chlorine in pesticide solutions was followed by microtitration; and the formation of degradation products, by gas chromatography. Aldrin and dieldrin were highly resistant to alkaline hydrolysis, but readily split off chlorine under ultraviolet irradiation, particularly in alkaline solution. Endosulfan and some related compounds (endodione, endolactone) were susceptible to alkaline hydrolysis, while others (endoether, chlorendic acid) were resistant. These and other derivatives undergo a series of highly complex photodegradation reactions. The hexachloronorbornene derivatives in turn are susceptible to photolysis, depending on the substituents at the 2,3-positions. A hydroxymethylene group at one of these positions imparts high reactivity, whereas the presence of two carboxylic acid groups or an additional ring at this point confers stability.

72-1843. Faust, S. D.; Gomaa, H. M. (Dept. of Environ. Sci., Rutgers, The State Univ., New Brunswick, NJ). **Chemical hydrolysis of some organic phosphorus and**

carbamate pesticides in aquatic environments. *Environ. Lett. 3(3): 171-201; 1972.* (18 references)

Information on chemical hydrolysis of organophosphates and carbamates is reviewed, along with consideration of the toxic effects of hydrolysis products. Several mechanisms have been reported for the hydrolysis of organophosphates. In general, compounds having an oxygen atom in the P group hydrolyze faster than those having a sulfur atom. Electron-withdrawing groups substituted at the R′ position facilitate hydrolysis. Different products can be obtained depending upon pH; in general, the organophosphates are more stable in an acidic medium. Compounds with higher activation energy requirements tend to be more persistent at temperatures of the natural environment (0-20°C). Carbamates undergo alkaline hydrolysis to a hydroxy compound, an amine, and CO_2 quite readily. The more widely used carbamates, however, are fairly stable at the neutral pH of natural waters. It can be seen that these substitutes for the "persistent" organochlorines and their metabolites can remain in the environment long enough to cause hazardous effects. Several breakdown products have been shown to be at least as toxic as the parent compound.

72-1844. Vermeer, K.; Armstrong, F. A. J. (Canadian Wildlife Serv., Edmonton, Alberta, Canada). **Mercury in Canadian prairie ducks.** *J. Wildl. Manage. 36(1): 179-182; 1972.* (6 references)

Tissues of 190 ducks, obtained during hunting season on the Canadian prairie, were analyzed for mercury. On the basis of two birds tested, common mergansers had the highest residues (over 0.5 ppm in breast muscle). Residues in livers were about 2.9 times higher than in breast muscles, and a fixed ratio was found for residues of these tissues. Residues in the primary feathers of adult pintails, but not immature pintails, were also found to correlate with residues in breast muscle. Exposure to seed dressings and contaminated fish was assumed to be responsible for the levels of mercury.

72-1845. Jefferies, D. J. (The Nature Conservancy, Monks Wood Exp. Sta., Abbots Ripton, Huntingdon, England). **Organochlorine insecticide residues in British bats and their significance.** *J. Zool. 166: 245-263; 1972.* (39 references)

Organochlorine determinations in 30 bats of 5 species taken from a locality in East Anglia from 1963 to 1970 showed that 100% of the livers contained *p,p′*-DDE; 82% contained *p,p′*-DDT; and 29% contained dieldrin. A recent, continuous exposure to DDT or a slow rate of metabolism of the compound to DDE was suggested by the quantity of DDT in tissue relative to total DDT-type material. Total DDT was highest in liver samples taken at the end of hibernation, possibly due to the low fat depot at this time. In a laboratory study of 30 bats, an approximate acute oral LD50 of 63 mg/kg was obtained. The day before death an intermittent, muscular trembling began and gradually become more violent. Levels between 13 and 45 ppm in the liver represent the upper limits for survival in field bats and the lower limits of laboratory bats succumbing to DDT poisoning. The rate of metabolism in laboratory-housed bats was slow relative to the rate in a passerine bird.

72-1846. Fujita, M.; Iwashima, K. (Dept. of Public Health Pharmaceutics, Inst. of Public Health, Tokyo, Japan). **[Residual mercury compounds in rice.]** *Koshu Eiseiin Kenkyu Hokoku (Bull. Inst. Pub. Health Tokyo) 19(3): 212-220; 1970.* (33 references) (Japanese)

An investigation of the distribution and chemical state of residual mercury in rice revealed that the mercury content of rice bran is higher than that of polished or unpolished rice. Separation of rice bran components under mild conditions gave a fat and lipid fraction, three protein fractions, and cellulose. The highest mercury content was found in the protein fraction extracted with 0.05 *N* NaOH. This fraction was examined by gel filtration, with or without prior digestion with pronase. Mercury in the separated fractions was determined by neutron activation analysis, and the mercury compound identified by thin-layer chromatography. The results revealed that the majority of the residual mercury present in rice is firmly bound to the rice bran protein and exists in a chemical form closely resembling phenylmercury.

72-1847. Mattheis, I.; Arfert, H.; Jung, N. (Hygiene-Institut Neustrelitz, DDR). **Zum Gehalt von DDT und γ-HCH in Wildvogeleiern. [The DDT and lindane contents of wild bird eggs.]** *Nachrichtenbl. Pflanzenschutzdienst DDR 25(10): 207-211; 1971.* (19 references) (German)

The DDE+DDT and lindane contents of 113 eggs collected from 14 species of birds in southeastern Mecklenburg were determined by thin-layer chromatography. Lindane was not detected except in two duck egg specimens. Total DDT content ranged from undetectable in the lapwing eggs examined through the 1-ppm range for duck and crow species; 2 ppm for buzzard, seagull, and two titmouse species; 4 ppm for cuckoo and grey goose; 5 ppm for great tit and blackbird; and 6 ppm for flycatcher. The anticipated correlations between dietary habits and insecticide content were found with a few exceptions, e.g., the marked deviations between titmouse species. The high DDT level in flycatcher eggs is explained both by the fact that their diet consists exclusively of insects and by the fact that some of the eggs were collected from an area recently sprayed with DDT for control of the May beetle. The unexpectedly low (undetectable) DDT level in the lapwing eggs may be attributable to the fact that these eggs were collected from a nature preserve area. The presence of DDT in the eggs of other bird species collected from the nature pre-

serve indicates that these birds obtained at least part of their food from other areas, where the DDT content of plants and insects was higher; thus, the establishment of nature preserves alone is not sufficient to insure the survival of birds.

72-1848. Beitz, H.; Seefeld, F.; Hartisch, J.; Heinisch, E. (Institut fuer Pflanzenschutzforschung Kleinmachnow, Biologische Zentralanstalt Berlin der Deutschen Akademie der Landwirtschaftswissenschaften zu Berlin, DDR). **Zur Aufnehmbarkeit von DDT durch Kulturpflanzen aus DDT-kontaminierten Boeden und die sie beeinflussenden Faktoren. [Uptake of DDT from DDT-contaminated soils by crops and the factors influencing uptake.]** *Nachrichtenbl. Pflanzenschutzdienst DDR 25(11): 229-234; 1971.* (19 references) (German)

A series of studies on DDT residues in food plants was performed by the regional hygiene institutes in East Germany. At DDT soil residues of 0.7-1.9 ppm, the residues in spinach leaf were between 0.07 and 0.3 ppm (60 days after planting). The residues in beet roots grown at similar soil DDT levels were between 0.08 and 0.6 ppm. Residues in spinach amounted to 0.03-0.1 ppm. In potatoes, grown in soil containing 1.3-2.3 ppm DDT, the DDT residues at the time of harvest ranged from 0.06 to 0.3 ppm. Generally, the below-ground parts of the plants contained higher DDT levels than the above-ground parts; therefore, vegetables in which the subterranean plant parts are eaten are likely to display higher DDT residues than those in which the leaves represent the edible portion. Factors which influence the uptake of DDT by crops include the DDT content of the soil, the soil type, the duration of the growth period, the fat content of the vegetables, and miscellaneous factors such as soil moisture, DDT formulation, and plant species. Since two-thirds of the soil samples investigated contained more than 0.1 ppm DDT, the possibility of crop contamination exists. In the case of crops used in producing baby foods and convalescent diets, in particular, it is necessary to make sure that the soil contains no more than 0.5 ppm DDT.

72-1849. Galley, D. J. (Dept. of Zool., Imperial College, London, S.W.7a, England). **Persistence of some organophosphorus insecticides in hen-house litter.** *Pestic. Sci. 3(1): 19-23; 1972.* (9 references)

Thin-layer chromatography was used to assess semi-quantitively the persistence of six organophosphorus insecticides in hen-house litter. Malathion disappeared within 4 hr of application. Carbofenthion was the most persistent. Four weeks after treatment the proportions of the applied doses extracted from the litter were: carbofenthion 46%, phorate 21%, diazinon 13%, coumaphos 7%, and fenitrothion 3%. (Author abstract by permission)

72-1850. Walker, A. (Natl. Vegetable Res. Sta., Wellesbourne, Warwick, England). **Availability of atrazine to plants in different soils.** *Pestic. Sci. 3(2): 139-148; 1972.* (20 references)

The concentrations of atrazine in the shoots of wheat plants growing in 12 different soils were directly proportional to the soil solution concentrations of herbicide estimated from slurry adsorption measurements. There was a marked discrepancy between the total uptake of herbicide and the amount theoretically supplied by mass-flow in response to transpiration. This discrepancy was less when plants were grown in nutrient solutions. In an experiment with one soil only, the half-life of atrazine was 22 days and when the solution concentration in this soil was corrected for this change, a much closer prediction of atrazine uptake could be obtained. The ways in which interactions between adsorption, breakdown, and transpiration rates may affect herbicide toxicity under field conditions are discussed. (Author abstract by permission)

72-1851. Findlay, E. (Dept. of Agr. and Fisheries, Agr. Scientific Serv., East Craigs, Edinburgh EH12 8NJ, Scotland). **Pesticide residues in foodstuffs in Great Britain. Pesticide residues in raspberries and strawberries.** *Pestic. Sci. 3(2): 169-174; 1972.* (5 references)

Over the period 1968/70, 147 samples of raspberries and 124 samples of strawberries were analysed for organochlorine insecticide residues. DDT was found in 30% of the samples but only one sample contained as much as 1.4 ppm. No residues were detected in the raspberries during the last year of the survey, which coincided with the discontinued use of DDT on this crop. 184 of the above samples were also analysed for residues of organophosphorus insecticides and the fungicide dichlofluanid. Malathion was found in one sample of raspberries. Approximately 40% of the strawberries contained detectable amounts of dichlofluanid and its metabolite with combined residues ranging from 0.1 to 7.5 ppm. Only two samples of raspberries contained residues of either dichlofluanid or its metabolite. It is considered that the residues of these various pesticides should not present a hazard to consumers. (Author abstract by permission)

72-1852. Smith, D. C.; Sandi, E.; Leduc, R. (Food and Drug Directorate, Dept. of Natl. Health & Welfare, Tunney's Pasture, Ottawa, Ont., Canada). **Pesticide residues in the total diet in Canada. II—1970.** *Pestic. Sci. 3(2): 207-210; 1972.* (4 references)

Composites of food representing the Canadian diet were analyzed for organochlorine and organophosphorus pesticides and arsenic. Thirteen organochlorine and three organophosphorus pesticides were found. Residues were consistently low and would contribute to the diet approximately 20 μg total pesticide residues and 30 μg arsenic per person per day. (Author abstract by permission)

72-1853. Poland, J. J.; Kissam, J. B.; Reed, J. K.; Barnett, B. D. (S. Carolina Agr. Exp. Sta., Clemson U., Clemson, SC 29631). **The uptake of DDT by domestic turkeys ranged on treated clay loam soil.** *Poultry Sci. 51(3): 1024-1026; 1972.* (10 references)

The accumulation of DDT in commercial turkeys and its persistence in soil over an 11-mo period is described. Turkeys were ranged for 20 wk on plots treated with 0, 11.2, 22.4, and 44.8 kg DDT/ha. Birds were sampled by slaughter at 4-wk intervals and mesenteric fat was analyzed. Even though the variation in residues was greater than anticipated in some instances, it is evident that even after 4 wk at the lowest DDT dosage that uptake was significant. The control birds in untreated plots, but adjacent to DDT plots, accumulated DDT above the average background level thus demonstrating the difficulty of confining this pesticide to a given area. (Author abstract by permission)

72-1854. Li, G.-C.; Felbeck, Jr., G. T. (U. of Rhode Island, Kingston, RI). **A study of the mechanism of atrazine adsorption by humic acid from muck soil.** *Soil Sci. 113(2): 140-148; 1972.* (32 references)

Adsorption isotherms obtained from studies on adsorption of atrazine on humic acid at various temperatures seem to conform to the Langmuir isotherm. On 200 mg of humic acid, 29.41 μM of atrazine was adsorbed at 25°C and about 7.07 μM was adsorbed at 3°C. The difference was apparently due to the number of sites available at the different temperatures. Results of studies using methylated humic acid and humic acid saturated with calcium indicated that the primary adsorption sites on humic acid are carboxyl groups and/or phenolic hydroxyl groups. A decrease in the amount of polar solvent on the humic acid appeared to stabilize the atrazine-humic acid complex.

72-1855. Yamane, V. K.; Green, R. E. (Dept. of Agron. and Soil Sci., U. of Hawaii, Honolulu, HI 96822). **Adsorption of ametryne and atrazine on an oxisol, montmorillonite, and charcoal in relation to pH and solubility effects.** *Soil Sci. Soc. Amer. Proc. 36(1): 58-64; 1972.* (28 references)

Ametryne was adsorbed to a greater degree than atrazine at pHs of approximately 3 and 7 on montmorillonite and Molokai surface and subsoils. The difference in adsorption on the subsoil was small. Adsorption isotherms were nearly linear on a log-log plot. The presence of charcoal, a common constituent of sugarcane soils in Hawaii, caused the higher adsorption to occur at the high pH, a reverse of the pattern observed without charcoal. Charcoal also increased adsorption of the triazines. It was evident from adsorption plots adjusted for temperature effects on solubility using relative concentrations that temperature-dependent solute-solvent affinity played an important role in triazine adsorption. Wetting agents used in ametryne commercial preparations did not change its adsorption characteristics.

72-1856. Igue, K.; Farmer, W. J.; Spencer, W. F.; Martin, J. P. (Dept. of Soil Sci. and Agr. Eng., U. of California, Riverside, CA 92502). **Volatility of organochlorine insecticides from soil: II. Effect of relative humidity and soil water content on dieldrin volatility.** *Soil Sci. Soc. Amer. Proc. 36(3): 447-450; 1972.* (7 references)

Volatilization of dieldrin, a representative weakly polar insecticide, from Gila silt loam was found to be dependent on soil water content and, indirectly, on relative humidity which affected soil water content, but not on rate of evaporation of water. When soil water loss was zero, dieldrin volatilization from moist soils was maximum. At less than 100% relative humidity, dieldrin volatilized at a decreasing rate as the soil water diminished, and at 2.8% moisture content no volatilization took place. At temperatures of 20° and 30°C, flow rates of 2 and 8 ml/sec, and dieldrin concentrations of 5 and 10 μg/g, the results always indicated that soil water content influences volatilization, probably by competing for adsorption sites. The "codistillation theory" does not appear to apply to volatilization of dieldrin from soils.

72-1857. Phillips, W. M.; Feltner, K. C. (Plant Sci. Res. Div., Agr. Res. Serv., U.S. Dept. of Agr., Hayes, KS 67601). **Persistence and movement of picloram in two Kansas soils.** *Weed Sci. 20(1): 110-116; 1972.* (10 references)

Persistence and movement of 4-amino-3,5,6-trichloropicolinic acid (picloram) were studied in silty clay loam soils located at Hays and Newton, Kansas. At Hays, picloram was applied to a Harney silty clay loam at rates of 2.24 and 3.36 kg/ha in 1963, 1964, 1965, 1966, and 1968 and at Newton to a Goessel (tentative) silty clay loam at rates of 1.12, 2.24, and 3.36 kg/ha in 1966. Fifteen-, 20-, or 30-cm increments of soil were taken from as deep as 2.4 m below the soil surface and bio-assayed with soybeans (*Glycine max* (L.) Merr. 'Clark 63') in 1968 and 1969. In both soils the highest concentration of phytotoxic residue remained near the soil surface, but differences between the two sites were evident. In the Goessel soil, phytotoxic residues were detected only near the surface. In the Harney soil, movement was uneven, but residues were detected as deep as 2.4 below the surface. Soil cracks probably contributed to penetration of small amounts of the herbicide to as much as 2.4 m below the surface. Total picloram remaining in both soils diminished with time. (Author abstract by permission)

72-1858. Swann, C. W.; Behrens, R. (Dept. of Agron. and Plant Genet., U. of Minnesota, St. Paul, MN 55108). **Trifluralin vapor emission from soil.** *Weed Sci. 20(2): 147-149; 1972.* (8 references)

Vapor of α,α,α-trifluoro-2,6-dinitro-*N*,*N*-dipropyl-*p*-toluidine (trifluralin) emitted from soils was collected in xylene and assayed by gas chromatography. Increasing

the trifluralin application rate in soil surface treatments from 0.56 to 4.48 kg/ha resulted in substantial increases in vaporization from soils during the 2 hr immediately after treatment. Increasing the spray volume from 2.34 to 300 kl/ha with water reduced the rate of trifluralin vapor emission by approximately half. When trifluralin was applied as a spray (2.34 kl/ha) and then incorporated into soils of varying moisture contents, 4 to 10 times more trifluralin volatilized in 12 hr than when it was percolated into air-dry soils with sufficient water to yield equivalent soil moisture contents. The techniques employed were not sufficiently sensitive to detect trifluralin vapor loss occurring more than 12 hr after treatment. The amount of trifluralin lost during the first 12 hr was less than 5% of that applied to the soil, even under the most favorable conditions for volatilization. (Author abstract by permission)

72-1859. Timmons, F. L.; Frank, P. A.; Demint, R. J. (Crops Res. Div., ARS, USDA, Laramie, WY). **Herbicide residues in agricultural water from control of aquatic and bank weeds.** *In: Agricultural Practices and Water Quality, T. L. Willrich and G. E. Smith, ed., Iowa State Univ. Press, Ames, Iowa, 1970, pp. 194-208.* (31 references)

Herbicide residues in water resulting from surface runoff are not large, and the total volume of water affected is small. Specific concentrations of herbicide in water are used for control of submersed weeds, but these generally dissipate rapidly. Granular formulations confine portions of the herbicides at the hydrosoil surface. Maximum levels of herbicides obtained experimentally from spraying ditch banks are unlikely to injure plants or produce significant residues in crop plants. Specific dissipation studies illustrate the various routes of dissipation, including volatilization in the case of aromatic solvents; sorption of water herbicides such as paraquat; and biological and chemical degradation. There is no substantial evidence of damage to crops by use of irrigation water from ditches treated with aromatic solvents; acrolein; copper sulfate; 2,4-D; dalapon; amitrole; 2,4,5-T; or silvex. Residues of only a few of the herbicides used have been found in crops.

72-1860. Le Grand, H. E. (U.S. Geological Survey, USDI, Raleigh, NC). **Movement of agricultural pollutants with groundwater.** *In: Agricultural Practices and Water Quality, T. L. Willrich and G. E. Smith, ed., Iowa State Univ. Press, Ames, Iowa, 1970, pp. 303-313.* (18 references)

Some of the geological conditions and hydrologic factors affecting the movement of pesticides and other agricultural pollutants in the ground are discussed. Pollutants are generally dissipated in the zone of aeration, which lies between the land surface and the water table. The chemical and biological character, texture, permeability, and thickness of the soil zone are determining factors in the degree to which decay, sorption, and dilution affect the travel of pollutants. The lack of concentration and the lack of continual application tend to prevent pesticides and fertilizers from moving through the aeration zone to the water table. Dumping large quantities of these chemicals on the ground, particularly where the zone of aeration is thin, could result in a pollution problem.

72-1861. Ragab, M. T. H.; Leefe, J. S. (Res. Sta., Canada Agr., Kentville, Nova Scotia, Canada). **Residues in soils and strawberries resulting from simazine applications.** *Can. J. Plant Sci. 52(2): 147-149; 1972.* (14 references)

Simazine applications that were effective in controlling weeds in strawberry plots were not found to produce residues in the fruit. Fruit from two separate harvests contained no detectable amounts of simazine. Fortified samples gave > 95% recoveries, and the limit of detection was 0.02 ppm. Soil samples taken from the most recent planting at 0 to 2.5 and 2.5 to 7.5 cm depths contained residues up to 0.47 ppm, and approximately 14% of the dosage remained in the soil 1 yr following treatment.

72-1862. Geisman, J. R. (Ohio Agr. Res. and Develop. Center, Wooster, OH). **Reduction of pesticides from horticultural food crops by processing.** *HortScience 7(3): 227-228; 1972.* (11 references)

Methods for reduction or removal of residues from horticultural food products were investigated. Studies on the preparation and processing of fruits and vegetables indicated that the use of a low-foaming wetting agent, warm water, agitation, and a high-pressure spray rinse are most effective for removal of insecticide or herbicide residues. However, the compound may still enter the food chain via the water supply. Ideally, the pesticide should be removed from the food chain altogether. The consumer may also remove the residues by washing or cooking. The removability of residues varies with chemical, timing of application, and plant species.

72-1863. Hernandez, E.; Martinez, R. M.; Martinez, M.; Terol, J. F. (Instituto de Agroquimica y Tecnologia de Alimentos, Valencia, Spain). **Proteccion de frutos citricos contra la putrefaccion mediante tratamientos con 1-(butilcarbamoil) benzimidazol-2-carbamato de metilo, (benomyl). Residuos del fungicida en los frutos tratados. [Protection of citrus fruits against spoilage by treating with methyl 1-(butylcarbamoyl)benzimidazole-2-carbamate (benomyl). Fungicide residues in treated fruits.]** *Rev. Agroquim. Tecnol. Aliment. 11(2): 249-259; 1971.* (6 references) (Spanish)

Benomyl has shown promise as a fungicide for oranges. Benomyl residues in whole orange, pulp, and peel were determined colorimetrically, following applica-

tion of an extensive cleanup procedure to remove interfering materials. The limit of detection was 0.2 ppm. Immediately after immersion of oranges for 4 min in a suspension containing 200 ppm of benomyl, the peel contained 2.4 ppm of benomyl, but no residues were detectable in the pulp. The residues in the peel decreased to 1.5 ppm after 30 days of storage at room temperature, and 1.3 ppm after storage for 28 days under refrigeration (0-3°C). No residues were detectable in the pulp at any time. No undesirable flavor or appearance of treated oranges was found in organoleptic tests.

72-1864. Montgomery, M.; Yu, T. C.; Freed, V. H. (Dept. of Agr. Chem., Oregon State U., Corvallis, OR 97331). **Kinetics of dichlobenil degradation in soil.** *Weed Res. 12(1): 31-36; 1972.* (7 references)

Samples of Willamette silt loam were treated with dichlobenil to give an initial concentration of 11 ppm and assayed for dichlobenil and its metabolites at 2-wk intervals during 38 wk in incubation at 6.7° and 26.7°C. At the lower temperature, a lag time of 8-10 wk intervened before degradation of dichlobenil began. Degradation then occurred according to a first-order rate law, which yielded a half-life of 28 wk. There was no lag time at the higher temperature, and the half-life was 19 wk. The activation energy found from the Arrhenius plots amounted to 3.57 kcal/mole, which is relatively low. The majority of the chemical loss was accounted for by formation of metabolites, principally 2,6-dichlorobenzamide.

72-1865. Kirkland, K.; Fryer, J. D. (Agr. Res. Council, Weed Res. Organization, Begbroke Hill, Yarnton, Oxford, England). **Degradation of several herbicides in a soil previously treated with MCPA.** *Weed Res. 12(1): 90-95; 1972.* (15 references)

Soil samples taken from plots treated with MCPA at 3.3 kg/ha twice a year (total, nine treatments) were examined for ability to degrade MCPA and related herbicides 6 mo after the last application of MCPA. The pretreated and untreated (control) samples were packed into tins and sprayed with solutions of MCPA, MCPB, dichlorprop, mecoprop, fenoprop, and dicamba at the rate of 3.3 kg/ha, which gave a concentration of about 3 ppm after mixing. Disappearance of herbicides was followed by the rapid root growth inhibition method, using cotton as the test species for MCPB and sorghum for the other herbicides. No MCPA was detected in soil pretreated with MCPA after 4 days of incubation, in contrast to the untreated soil, where residues were still detectable after 8 days, but not after 16 days. MCPB was degraded at the same rate as MCPA. Pretreatment of the soil with MCPA did not influence the rate of disappearance of dichlorprop, mecoprop, or dicamba. There was a large difference between the initial recoveries of applied fenoprop from pretreated and untreated soils, but the subsequent breakdown curves were similar. Indirect evidence suggests that β-oxidation of MCPB to MCPA did not occur to a significant extent in the soil surrounding the roots of the test plants or within the plants.

72-1866. Smith, A. E. (Canada Dept. of Agr., Box 440, Regina, Sask., Canada). **Persistence of trifluralin in small field plots as analyzed by a rapid gas chromatographic method.** *J. Agr. Food Chem. 20(4): 829-831; 1972.* (12 references)

A simple and rapid method is described for the gas chromatographic determination of trifluralin residues in soils using an electron-capture detector. Recoveries of 89 to 99% are obtained from three soil types treated at the 0.1- to 1.0-ppm level. Small field plots 18 X 18 cm in size were treated with trifluralin at rates equivalent to 1.4 kg/ha at two locations in Saskatchewan. After 5-mo growing season, the residues remaining in the 0- to 5-cm and 5- to 10-cm levels of soil were determined. Between 11 and 21% of the applied herbicide was found in the top 5 cm of the soil, while less than 2% of the trifluralin was detected at the 5- to 10-cm levels. (Author abstract reprinted by permission of the American Chemical Society)

72-1867. Lichtenstein, E. P.; Schulz, K. R.; Fuhremann, T. W. (Dept. of Entomol., U. of Wisconsin, Madison, WI 53706). **Movement and fate of Dyfonate in soils under leaching and nonleaching conditions.** *J. Agr. Food Chem. 20(4): 831-838; 1972.* (11 references)

The degradation and movement of ^{14}C-Dyfonate in different soil types was tested in the laboratory under leaching and nonleaching conditions, at different temperatures and various incubation times. Different apparatuses are described which had been especially designed for testing the movement of pesticidal chemicals in soils with and without water. Although Dyfonate did not move to an appreciable extent through an agricultural loam soil, its transport with water through soils was, to a large extent, a function of the soil type. Water, after its percolation through a silt loam, did not contain measurable amounts of the insecticide, while water after its percolation through a sandy loam soil was toxic to mosquito larvae and contained 4% of the dosage originally applied to the top soil layer. After degradation of ^{14}C-Dyfonate in soils, radiocarbon was lost from the soil by volatilization of the ethoxy moiety and partially by transport of the ring moiety with water. When no water was percolated the insecticide also degraded and moved into untreated soil, but to a lesser degree. (Author abstract reprinted by permission of the American Chemical Society)

72-1868. Smith, S.; Parr, J. F. (Soil and Water Conservation Res. Div., ARS, USDA, P.O. Drawer U, University Station, Baton Rouge, LA 70803). **Chemical stability of**

DDT and related compounds in selected alkaline environments. *J. Agr. Food Chem. 20(4): 839-841; 1972.* (13 references)

DDT was stable in soil treated with anhydrous ammonia (pH > 10.0) and in sterile, buffered, glass microbeads up to pH 12.0. The threshold pH for dehydrochlorination of DDT to DDE in microbeads was 12.5, with extensive conversion (>70%) at pH 13.0, where the amount of applied DDT unaccounted for increased from 20% at 140 hr to approximately 50% after 30 days, suggesting the formation of intermediates that were lost during extraction or not detectable by electron capture. Applied DDE was relatively stable in microbeads even at pH 13.0 where nearly complete recovery was obtained after 7 days. However, extended incubation to 28 days allowed a gradual disappearance of DDE with only 88 and 74% accounted for at pH 10.0 and 13.0, respectively, suggesting a time-dependent pH relationship for transformation under these conditions. Similar observations were observed in studying the effect of pH on the dehydrochlorination of DDD (TDE) to DDMU. While DDD was stable for extended periods at pH 10.0, it converted rapidly to DDMU at pH 13.0 and then tended to disappear with time. (Author abstract reprinted by permission of the American Chemical Society)

72-1869. Wilson, N. K.; Fishbein, L. (Natl. Inst. of Environ. Health Sci., P.O. Box 12233, Research Triangle Park, NC 27709). **Decomposition of tetrakis(*N,N*-diethyldithiocarbamato)selenium(IV).** *J. Agr. Food Chem. 20(4): 847-850; 1972.* (23 references)

The kinetics of the decomposition of tetrakis (*N,N*-diethyldithiocarbamato)selenium(IV) have been studied by proton magnetic resonance. The decomposition is first order in ethyl selanac and in water. Major decomposition products are *N,N,N'-N'*-tetraethylthiuram disulfide and selenium, obtained through selenious acid and *N,N*-diethyldithiocarbamic acid intermediates. The importance of decomposition of the intermediate *N,N*-diethyldithiocarbamic acid to diethylamine and carbon disulfide is greater at high water concentrations and low pH values. The activation thermodynamic parameters for the ethyl selenac decomposition are $\Delta G\ddagger =$ 19.9 kcal/mol, $\Delta H\ddagger =$ 17.5 kcal/mol, $\Delta S\ddagger = -8.2$ eu, and the Arrhenius activation energy, E_a = 18.1 kcal/mol. (Author abstract reprinted by permission of the American Chemical Society)

72-1870. Cheng, H.-M.; Yamamoto, I.; Casida, J. E. (Div. of Entomol., U. of California, Berkeley, CA 94720). **Rotenone photodecomposition.** *J. Agr. Food Chem. 20(4): 850-856; 1972.* (23 references)

Irradiation of rotenone in oxygenated methanol solution with ultraviolet light yields the following crystalline products: *O*-demethylrotenone; 6aβ-12aβ-rotenolone; rotenononе; and three acids (4,5-dimethoxysalicylic, rissic, and tubaic acids) which were methylated prior to their isolation. Rotenone photodecomposes on plant leaves exposed to sunlight and on glass surfaces irradiated with sunlight, ultraviolet light, or a sunlamp to yield at least 20 compounds, including *O*-demethylrotenone, 6′,7′-epoxyrotenone, 6aβ,12aβ- and 6aβ,12aα-rotenolones, 6′,7′-epoxy-6aβ,12aβ-rotenolone, 6a,12a-dehydrorotenone, and rotenononе. Methods of synthesis are described for the new *O*-demethyl- and 6′,7′-epoxy derivatives of rotenone. In the determination of residues in or on crops treated with rotenone, it is important to determine the major toxic photodecomposition product, 6aβ,12aβ-rotenolone, as well as rotenone. (Author abstract reprinted by permission of the American Chemical Society)

72-1871. McGuire, R. R.; Zabik, M. J.*; Schuetz, R. D.; Flotard, R. D. (Dept. of Entomol., Pestic. Res. Center, Dept. of Chem. and Inst. of Biol. and Med., Michigan State U., East Lansing, MI 48823). **Photochemistry of bioactive compounds. Photochemical reactions of heptachlor: kinetics and mechanisms.** *J. Agr. Food Chem. 20(4): 856-861; 1972.* (8 references)

The product formation, reaction kinetics, and mechanisms of the photolysis of heptachlor (1,4,5,6,7,8,8-heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene, I) have been investigated. Photolysis at 253.7 nm in hydrocarbon solvents yields two olefinic monodechlorination isomers (II, III) ($\Phi = 0.025$); at 300 nm in acetone, a 2 + 2 cycloaddition or cage compound (IV) is the exclusive product ($\Phi = 9.35 \times 10^{-5}$ based on total absorption of energy by acetone); and in mixed cyclohexane/acetone solutions at 300 nm, IV and a C-1 cyclohexyl adduct (V) are formed *via* a triplet and allylic free radical, respectively. A cage opening reaction of IV occurs at 200 nm to yield I ($\Phi = 0.195$). Photodechlorination to yield II and III occurs *via* excitation of the 5,6 double bond, most probably through a singlet state; while formation of IV and V occurs *via* a triplet mechanism involving the 2,3 double bond. A kinetic mechanism and specific rate constants are reported. (Author abstract reprinted by permission of the American Chemical Society)

72-1872. Paulson, G. D.; Docktor, M. M.; Jacobsen, A. M.; Zaylskie, R. G. (U.S. Dept. of Agr., Entomol. and Animal Sci. Div., ARS, Metabolism and Radiation Res. Lab., Fargo, ND 58102). **Isopropyl carbanilate (propham) metabolism in the chicken: balance studies and isolation and identification of excreted metabolites.** *J. Agr. Food Chem. 20(4): 867-876; 1972.* (17 references)

Leghorn hens were given a single oral dose of either isopropyl-(2-^{14}C) carbanilate (A) or isopropyl carbanilate-[phenyl-^{14}C(U)] (B). Total carbon-14 excreted in the urine accounted for 79.8% of the activity given as A and 87.0% of the activity given as B during the 48-hr collection period; the fecal elimination ac-

counted for 6.4 and 7.0% of carbon-14 given as A and B, respectively. When A was given, 6.7% of the carbon-14 was expired, whereas only trace amounts of expiratory carbon-14 were detected when B was given. Carbon-14 remaining in the hens 48 hr after dosing accounted for 1.2 and 1.5% of the activity given as B and A, respectively. Eggs collected for 12 days after dosing contained 0.075% of the carbon-14 given as a single oral dose of B. Urinary metabolites were identified as the glucuronide conjugate of isopropyl 4-hydroxycarbanilate (I), *p*-aminophenyl sulfate, the sulfate ester of isopropyl 3-methoxy-4-hydroxycarbanilate, the sulfate ester of isopropyl 4-hydroxycarbanilate (VI), the 3-sulfate ester of isopropyl 3,4-dihydroxycarbanilate (VII), an incompletely identified conjugated form of isopropyl 3,4-dihydroxycarbanilate, and the sulfate ester of isopropyl 3-hydroxycarbanilate. The feces contained I, VI, VII, and isopropyl 4-hydroxycarbanilate. (Author abstract reprinted by permission of the American Chemical Society)

72-1873. Childers, A. B.; Carson, C. A.; Landmann, W. A. (Depts. of Vet. Pub. Health and Biochem., Texas A & M U., College Station, TX 77843). **DDT residue depletion in sheep using dietary energy restriction and administration of glucagon.** *J. Agr. Food Chem. 20(4): 878-880; 1972.* (28 references)

DDT residue depletion in sheep was studied using dietary energy restriction and treatment with glucagon. After 104 days DDT residues had decreased as follows: control, 12.8%; dietary energy restriction, 39%; dietary energy restriction + glucagon (1 mg/100 kg), 51.2%. Glucagon caused a 14% decrease in blood glucose after 6 days of administration. Dietary energy restriction and glucagon treatment produced a statistically significant ($p < 0.05$) reduction in DDT residues. However, glucagon did not produce a statistically significant ($p < 0.05$) reduction over dietary energy restriction alone. Neither dietary energy restriction nor treatment with glucagon appears to offer a practical solution to the problem of DDT residues in meat animals. (Author abstract reprinted by permission of the American Chemical Society)

72-1874. Getzendaner, M. E.; Corbin, H. B. (Ag-Organics Dept., Dow Chemical U.S.A., Midland, MI 48640). **Residues on apples and pears from use of Plictran miticide.** *J. Agr. Food Chem. 20(4): 881-885; 1972.* (4 references)

Apples and pears from trees in several experiments in various sections of the United States, sprayed with Plictran miticide, were analyzed for residues of total and organic tin. Inorganic tin was calculated as the difference between total and organic tin. Residue levels were similar on the two fruits and a maximum of less than 2 ppm of organotin was found, calculated as tricyclohexyltin hydroxide (Cy_3SnOH) on the day of the last of four applications. Organotin decreased to about half of the initial value in 3 to 5 wk. Most of the residue of total tin remaining 4 wk after spraying was organic tin, and most of that was the active ingredient Cy_3SnOH. Nearly all the residue was on the peel of the fruit, and up to half of this was removed with a cold water wash. (Author abstract reprinted by permission of the American Chemical Society)

72-1875. Juszkiewicz, T.; Stec, J.; Radomanski, T.; Trebicka-Kwiatkowska, B. (Zaklad Farmakologii Instytutu Weterynarii, Pulawy, Poland). **Pozostalosci insektycydow polichlorowych w siarze i mleku kobiet po porodzie. [Residues of organochlorine insecticides in the colostrum and milk of women after delivery.]** *Pol. Tyg. Lek. 27(17): 616-619; 1972.* (17 references) (Polish)

Analysis (gas-liquid and thin-layer chromatography) of milk and milk lipids obtained on the 3d, 5th, and 7th day after delivery from 32 women from a rural area revealed the following mean concentrations (ppm) of DDT + DDE + DDD (TDE): in milk; 0.22 ± 0.03 on 3rd, 0.25 ± 0.02 on 5th, 0.29 ± 0.98 on 7th day; in milk fat; 11.48 ± 1.15, 10.62 ± 0.98, and 11.29 ± 0.95, respectively. The DDT:DDE:DDD ratio was 40%:59%:1%. DDT and DDE were found in all; and TDE and BHC (av. mean 0.003 ppm), in 53% of women. Comparison of residue values in milk fat of women with those in fatty tissue in the general population (previous study) and those in milk fat of cows revealed that the mean concentration of DDT and metabolites (11.30 ppm) and BHC (0.11 ppm) in human milk fat corresponds to that found in human fatty tissue (11.36 and 0.13 ppm, respectively) but is significantly higher than in cow's milk fat (0.61 and 0.05, ppm respectively). It is stressed that these residues in human milk exceed (by 9 times) the accepted (FAO, WHO) tolerance limits.

72-1876. Bogusz, M. (Instytut Ekspertyz Sadowych, Krakow, Poland). **Insektycydy polichlorowe: o potrzebie realnej oceny ryzyka. [Organochlorine insecticides: the need for a realistic evaluation of risk.]** *Pol. Tyg. Lek. 27(17): 637-640; 1972.* (19 references) (Polish)

In a discussion and brief review, the alarm expressed by nonprofessionals and the lay press on the danger of DDT is downgraded, and the continued, rational and careful usage of organochlorine insecticides (OCI) recommended. Three points are stressed. First, exposure to constant daily dosages of OCI does not lead to an unlimited concentration in the body: after approximately 1 yr, a dynamic equilibrium is established, and the concentration of pesticides remains proportional to daily dose. Second, no deleterious clinical effect was discovered in volunteers exposed to dosages 100-200 fold larger than average. Third, the increased activity of microsomal enzymes of the liver resulting from OCI ex-

posure is an adaptive process and is not to be considered as evidence of liver damage. The following concentrations of "total DDT" (DDT + DDE) among the Polish population have been reported: in fatty tissue (ppm), 13.4 and 11.36; human milk (mg/l.), 0.27 and 0.40; lipid fraction of milk (ppm), 12.87; blood (mg/l.) male, 0.033; female, 0.057.

72-1877. Ware, G. W. (Dept. of Entomol. U. of Arizona, Tucson, AZ). **Pesticide drift . . . dust vs. spray.** *Progr. Agr. Ariz. 24(1): 10-11; 1972.*

In connection with the removal of most dust formulations from the list of agricultural insect control suggestions for 1972 by the University of Arizona Department of Entomology, some facts concerning dust and spray drift are reviewed. In general, dusts drift more than sprays and deposit less pesticide on the target crop. The use of dusts is now generally restricted to dense foliage in orchards or to home and garden applications. The fall-rate of small particles depends much more on their size than on their density or shape, and sprays have the advantage of being able to be made coarse. Average diameters of carriers for dust formulations are in the 10-μ range, whereas in sprays, droplets of 50-300 μ are usually found. The longer the airborne time of a particle, the greater its opportunity to drift. Whereas a medium aircraft spray droplet (150 μ diameter) can drift 22 ft while falling 10 ft in a 3-mph wind, a 10-μ dust particle can drift 4436 ft. Thus the greater safety of sprays is assured.

See also **72-1813 72-1821 72-1887 72-1895 72-1928 72-1959 72-1960 72-1961 72-1982 72-2014 72-2017 72-2018 72-2019 72-2031 72-2032 72-2033 72-2037**

72-1878. Hunter, J.; Maxwell, J. D.; Stewart, D. A.; Williams, R.; Robinson, J.; Richardson, A. (Med. Res. Council Group on Metabolism and Haemodynamics of Liver Disease, King's Coll. Hosp. and Med. Sch., London, SE5, England). **Increased hepatic microsomal enzyme activity from occupational exposure to certain organochlorine pesticides.** *Nature (London) 237(5355): 399-401; 1972.* (19 references)

Hepatic microsomal enzyme activity was followed indirectly in control subjects and workers from a plant manufacturing aldrin, dieldrin, and endrin; measurements of urinary D-glucaric acid were taken and related to organochlorine blood levels. Office workers from the Dutch plant had D-glucaric acid levels in the range of British controls. Average values in subjects who had worked only with aldrin and dieldrin were not significantly different from those of the controls. A significant increase in D-glucaric acid excretion was observed in men employed in the manufacture of endrin alone and in most of those who had been exposed to all three compounds. There appeared to be no correlation with HEOD blood levels, which are a measure of pesticide concentration, and D-glucaric acid excretion. Levels of *p,p'*-DDE showed a significant inverse correlation with excretion of D-glucaric acid, further suggesting enzyme induction in employees exposed to endrin.

72-1879. Wolverton, B. C.; Richter, R.; Lefstad, S. (Air Force Armament Lab., Eglin AFB, FL 32542). **Organophosphorus decontaminant.** *Technol. Util. Ideas 70's Beyond, Proc. Spec. AAS (Amer. Astronaut. Soc.)/AIAA Tech. Event, 1970 (AAS Sci. Technol. Ser. v26, 1971) pp. 7-13.* (9 references)

An organophosphorus insecticide decontaminating solution, suitable for use on and around aircraft and spray equipment, converts the insecticides to water-soluble products of low toxicity to fish. The solutions, containing 12.5% or 25% monoethanolamine (MEA) dissolved in dipropylene glycol monomethyl ether (DPGME), were mixed with insecticide standard solutions at the rate of 10 parts decontaminant to one part insecticide. After the desired reaction time had elapsed, unchanged pesticides were extracted from the mixture with hexane and analyzed by gas chromatography. The more concentrated MEA solution was used for decontamination of malathion, dichlorvos, and fenthion, while the less concentrated solution was used in the case of naled to prevent the excessive temperature rise caused by the isothermal reaction. Most of the naled in solution was destroyed within 30 min, and most of the malathion in 60 min. About half of the fenthion remained unchanged after exposure to 25% MEA solution for 240 min. A high degree of effectiveness in destroying the insecticide components which are toxic to fish was recorded in bioassays, and the decontaminating solutions were not corrosive to aluminum. The reaction of MEA with organophosphate insecticides consists of a nucleophilic displacement reaction at the phosphorus.

72-1880. Snyder, R. D. (Dept. of Pediatrics, U. of New Mexico Sch. of Med., Albuquerque, NM 87106). **The involuntary movements of chronic mercury poisoning.** *Arch. Neurol. (Chicago) 26(4): 379-381; 1972.* (21 references)

Additional findings in the members of the New Mexico family poisoned by mercury ingested via pork from hogs fed mercury-treated seed grain are presented. Four of the patients in this family displayed involuntary movements as outstanding symptoms. The first victim, an 8-yr-old girl, initially developed ataxia and agitation. Neurological degeneration advanced to the comatose state, and no subsequent involuntary movements occurred. Initial symptoms in the second victim, a 13-yr-old boy, were also ataxia and agitation, followed by blindness and almost constant choreiform movements in face and distal extremities as well as athetoid movements involving neck, shoulders and tongue. Initial symptoms in the third victim, a 20-yr-old woman, were grimacing, athetoid movements of the neck and upper extremities, and ataxia of the upper and lower extremities. A baby born to the mother of these children 6 mo after the ingestion of the poisoned meat developed a dark skin coloration within the first minute of life, accompanied by tremulous movements of the face and extremities. The movements could be stopped by holding the baby. An electroencephalogram recorded on the day after birth was apparently normal. The movements disappeared over the first weeks of life, but neurological impairment progressed. Peculiar involuntary movements have also been recorded in other instances of mercury poisoning, and their unexplained occurrence should suggest this diagnosis.

72-1881. Kaye, S. (Inst. of Legal Med., School of Med., Puerto Rico). **Patterns of poisoning in Puerto Rico.** *Bol. Asoc. Med. P. R. 62(1): 18-21; 1970.* (2 references)

Of the 84 fatal poisoning cases detected at autopsy by the Institute of Legal Medicine, parathion or other organophosphate compounds accounted for 27, or nearly one-third. Nine of these cases were in females, and 18 in males. Five were classified as accidental, 19 as suicidal, and 9 could not be classified. Parathion was the most common agent used for suicide by males, and barbiturates by females. These figures compare with 19 fatalities due to parathion poisoning out of 60 poisoning deaths in 1967, 30 out of 69 in 1966, 31 out of 66 in 1965, and 24 out of 56 in 1964. This decline in the fraction of poisoning deaths due to parathion was accompanied by a rise in the fraction due to morphine derivatives and barbiturates.

72-1882. Damluji, S. F.; Tikriti, S. (Dept. of Med., Med. Coll., Baghdad U., Baghdad, Iraq). **Mercury poisoning from wheat.** *Brit. Med. J. 1(5803): 804; 1972.* (2 references)

In a Letter-to-the-Editor, a recent outbreak of

poisoning from Granosan M (ethylmercury *p*-toluene sulfonamide) in Iraq is reported. Poisoning occurred among farmers using wheat dressed with this fungicide. More than 5500 persons were hospitalized, and 280 died. A similar but smaller outbreak occurred in Iraq in 1961. Physicians with experience in similar cases, and particularly with experience in their treatment, are requested to contact the authors.

72-1883. Lyle, C. D. (Author address not given). **Quick action saves heifers from pesticide poisoning.** *Hoard's Dairyman 116(15): 856-857; 1971.*

A call from a farmer's wife reporting unusual behavior disturbances in four heifers seemed to the responding veterinarian to be indicative of poisoning. The morning before the disturbances occurred, the farmer had planted a field to corn, using an organophosphate and an organochlorine pesticide to protect the crop. The wagon used for hauling the supplies to the field had been parked in the yard where the heifers were held, and the heifers licked the wagon bed. Symptoms developed shortly thereafter, progressing from disorientation, muscular twitching, and spasms to hemorrhagic diarrhea, salivation, and gnawing, as well as difficulty in breathing and apparent abdominal pain. Atropine sulfate and chloral hydrate were administered until the symptoms were brought under control. By the following day, the condition of the heifers was normal, except for broken teeth and skin wounds due to the chewing and fighting.

72-1884. Karkos, J. (Pracownia Neuropatologii Instytutu Psychoneurologicznego, Pruszkow, Poland). **Obraz neuropatologiczny encefalopatii talowej. [Neuropathological findings in thallium encephalopathy.]** *Neurol. Neurochir. Pol. 21(6): 911-915; 1971.* (10 references) (Polish)

Ingestion of half a tablespoon of rat poison containing thallium by a 13-yr-old girl (suicide attempt) resulted in death 21 days later. Initial symptoms included severe pain in abdomen and lower extremities, loss of hair, constipation, elevated temperature followed by several episodes of obnubilation with accelerated heart beat, pulsating cervical artery, and labored, shallow breathing. Examination of brain tissue on autopsy revealed generalized degeneration of ganglion cells, damage to axons, and disintegration of myelin sheaths with relatively slight glial reactions. The changes were nonspecific and not localized in definite areas. Noteworthy was the fatty degeneration of ganglion cells (despite the young age of the patient), severe swelling of oligodendroglia, sieve-like condition of basal ganglia, and strong susceptibility of the calcar cortex to lesions.

72-1885. Biskind, M. S.; Mobbs, R. F. (Westport, CT). **Psychiatric manifestations from insecticide exposure.** *J. Amer. Med. Ass. 220(9): 1248; 1972.* (4 references)

In a Letter-to-the-Editor, the similarities between an epidemic of unknown etiology reported in a previous letter and several known insecticide poisoning incidents in hospital personnel are pointed out. The epidemic described represented manifestations of neuromyasthenia, primarily among hospital personnel, at Lackland Air Force Base, Texas. Principal symptoms included acute gastroenteritis, myalgia, and lethargy, and, in 18 of the 221 cases, encephalopathy. The authors point out that insect and rodent extermination procedures are performed quite frequently in most hospitals; in many cases, the housekeeping staff is not informed concerning the nature of the agent, and the professional staff may not even know that insecticides have been applied. Whereas organophosphates such as diazinon and carbamates such as propoxur decompose rather rapidly out of doors, they are more persistent indoors, where shielded from effects of sun and rain. The authors were consulted in several recent episodes in insecticide poisoning among hospital personnel. In one, outbreaks of nausea, diarrhea, headaches, confusion, and myalgia suggested insecticide poisoning to a secretary who had had a previous experience with diazinon. Outbreaks subsided when treated areas were washed with detergent.

72-1886. Pierce, P. E.; Thompson, J. F.; Likosky, W. H.; Nickey, L. N.; Barthel, W. F.; Hinman, A. R.* (845 Central Ave., Albany, NY 12206). **Alkyl mercury poisoning in humans. Report of an outbreak.** *J. Amer. Med. Ass. 220(11): 1439-1442; 1972.* (14 references)

Three members of a family of nine that ingested mercury-contaminated [fungicide] pork became ill with classic symptoms of methylmercury poisoning and rapidly progressed to coma or semicomatose states. Treatment with dimercaprol (BAL in Oil) and *N*-acetyl-*d,l*-penicillamine was not associated with significant immediate clinical improvement, although both drugs appeared to increase urinary excretion of mercury. All three patients improved considerably but still remain severely impaired. A case of congenital methylmercury poisoning also resulted from the outbreak. To our knowledge, this is the first known instance in the United States of methylmercury poisoning resulting from ingestion of mercury-contaminated meat. (Author abstract by permission)

72-1887. Smith, Jr., W. M.; Ledbetter, J. O. (Rohm and Haas Co., Deer Park, TX, and Environ., Eng., Coll. of Eng., U. of Texas, Austin, TX 78712). **Hazards from fires involving organophosphorus insecticides.** *Amer. Ind. Hyg. Ass. J. 32(7): 468-474; 1971.* (9 references)

An investigation was undertaken to define the toxic hazards of fires involving organophosphorus insecticides. Solutions of malathion and of parathion in a variety of solvents were burned, and the fire gases and residues were analyzed. Most of the insecticide present was destroyed by decomposition or combustion during

the fire. Some polymerization products were found. Distilled vapors of the insecticides escaping the fire were considerably diluted. A method for estimating the potential concentrations and hazards for a given situation is described. (Author abstract by permission)

72-1888. Anonymous. **Hexachlorocyclohexane, *gamma* isomer–lindane.** *Amer. Ind. Hyg. Ass. J. 33(1): 56-59; 1972.* (17 references)

The toxic properties of lindane and information regarding emergency treatment and hygienic standards and practices associated with its use are reviewed. Inhalation of lindane causes severe headaches, nausea, irritation of the eyes, nose and throat, and sometimes an allergic response; repeated exposure does not result in an increase in blood levels of the compound. Absorption through the skin can result in much higher blood levels of lindane, and 50 mg/kg body wt is considered a dangerous cutaneous level for higher animals. Acute oral doses causing ill effects in man seem to be quite varied. The acute oral LD50 dose for higher animals is about 125 mg/kg body wt. Gross symptoms of poisoning include tremors, ataxia, convulsions, and prostration. No direct field methods are reported for evaluation of exposure. Laboratory methods include IR and GLC analysis of air samples and chromatographic analysis of urine and blood. Control of work area concentrations to 0.5 mg/m^3 of air is necessary to avoid physiological effects on workers. Above this concentration, respiratory protective devices are recommended. Thorough washing of exposed clothing is necessary, and eyes should be protected with a facepiece or goggles. Emergency treatment requires immediate washing of exposed areas and medical attention. Gastric lavage and saline cathartic may be used. Convulsions can be controlled with sedatives, sometimes administered with calcium gluconate. Epinephrine should not be used.

72-1889. Savitt, L. E. (Gardena, CA). **Contact dermatitis due to benomyl insecticide.** *Arch. Dermatol. 105(6): 926-927; 1972.*

In a Letter-to-the-Editor, findings in seven cases of contact dermatitis following exposure to benomyl insecticide are reported. All of the cases involved women of Japanese ancestry employed in debudding or cutting carnations. In plastic greenhouses where temperature and humidity were high, the eruptions appeared on the dorsum of the hands and the forearms and sometimes in other areas not covered by clothing. The eruptions occurred only after second spraying of benomyl and were not seen in 10 men and women of Mexican ancestry performing similar work. The eruptions, consisting of redness and edema, cleared after approximately 3 wk of topical treatment with aluminum sulfate compresses and systemic administration of chlorpheniramine, prednisolone, corticotropin, and calcium. Patch tests in affected patients were positive; patch tests in controls were negative, except when UV irradiation was applied after benomyl exposure in a Japanese medical assistant. Urticaria and subungual hemorrhages were seen in two patients during a second eruption episode, but could not be positively correlated with benomyl contact. Inquiries to other agencies revealed that the only other confirmed case of benomyl allergy involved bilateral conjunctivitis in an agricultural research worker.

72-1890. Matsunaga, H. (Natl. Inst. of Genetics, Yata, Mishima-shi, Shizuoka-Prefecture, Japan). **[Environmental pollution and heredity: toward protecting the health of offspring.]** *Iden (Heredity) 26(6): 53-57; 1972.* (Japanese)

The problem of environmental pollution is particularly serious in Japan due to the small size and high population density of that country, as well as to its tremendous rate of industrial development in recent years. One area in which imperceptible threats to man exist is the area of mutagenesis. Mutagenic substances to which various population segments may be exposed include some pesticides. Ethyleneimine derivatives, used as insecticides and soil conditioners, captan, and maleic hydrazide have revealed a mutagenic potential for man. Testing for mutagenicity prior to registration of agricultural chemicals is one of the steps which can be taken to prevent contamination of the gene pool.

72-1891. Sandifer, S. H.; Keil, J. E.; Finklea, J. F.; Gadsden, R. H. (Ind. Med. and Surg., P. O. Box 7151, Ft. Lauderdale, FL 33304). **Pesticide effects on occupationally exposed workers: a summary of four years observation of industry and farm volunteers in South Carolina.** *Ind. Med. Surg. 41(5): 9-12; 1972.* (4 references)

Morbidity and mortality did not differ significantly between control volunteers and 120 volunteers who were occupationally exposed to a number of pesticides. However, a larger and more varied sampling of the population is needed to find slight differences in these parameters. No overt toxicity was revealed, but significant differences were noted in systolic blood pressure (but not catecholamines), serum cholesterol, ChE, white blood cells, hemoglobin, SGPT, and hematocrit. DDT levels were measured as an indicator of general exposure to pesticides.

72-1892. Hashimoto, Y. (Sect. of Soil Pestic., Environ. Agency, Chiyoda-ku, Tokyo, Japan). **[Avian toxicities of pesticides. Part 2. Fungicides and herbicides.]** *Kongetsu No Noyaku (Agr. Chem. Mon.) 16(4): 76-79; 1972.* (Japanese)

Data are presented concerning the toxicities of fungicides and herbicides to birds, in comparison to their mammalian toxicities. The group of compounds of high

toxicity to mallard ducks or quails (LD50 less than 30 mg/kg) includes endrin, diazinon, parathion, DDVP, and fenitrothion. Only two of these compounds, endrin and parathion, have high mammalian toxicity. The moderately toxic group (LD50 in birds, 30-300 mg/kg) includes aldrin, benzoepin, DNOC, and dimethoate; of these, benzoepin and DNOC are highly toxic in mammals. The group of chemicals of low toxicity to birds (LD50 more than 300 mg/kg) includes kelthane, sodium arsenate, DDT, carbaryl, and heptachlor, of which sodium arsenate and carbaryl are highly toxic in mammals. Aldrin, DNOC, and heptachlor are highly toxic to small fish. The inclusion of at least one bird species in the required testing of a pesticide prior to registration might help to prevent the poisoning of wild birds due to pesticide use.

72-1893. Hashimoto, Y. (Sect. of Soil Pestic., Environ. Agency, Chiyoda-ku, Tokyo, Japan). **Avian toxicities of pesticides. Part 3. Biologically concentrated pesticides and their effects; responses of wild birds to pesticide application.** *Kongetsu No Noyaku (Agr. Chem. Mon.) 16(5): 68-70; 1972.* (Japanese)

Recent findings regarding the toxicity of insecticides to birds, particularly the effects of concentration in the food chain, are reviewed. Evidence for biological concentration is indirect, consisting of the fact that insecticide (especially chlorinated hydrocarbon) residues are higher in insects than in plants, still higher in fish and small mammals, and highest in predatory birds. Such pesticides accumulate primarily in the liver and fat of birds and include DDT and its metabolites as well as BHC isomers in Japan. Apparently, when the organochlorine residues in the brain exceed a certain level, the bird dies. A statistical relationship exists between the quantity of pesticide ingested and the residues in the fatty tissues of birds, as well as the residues in the egg. High residue levels in the egg correlate with eggshell thinning. In the United States, application of heptachlor to meadows and forests resulted in a decrease in the number of individuals and species of birds. The population levels were apparently restored after 1 yr, but by this time the population of the target insect had also returned to its initial level.

72-1894. Hashimoto, Y. (Sect. of Soil Pestic., Environ. Agency, Chiyoda-ku, Tokyo, Japan). **[Avian toxicities of pesticides. Part 4. Effects on domestic fowl.]** *Kongetsu No Noyaku (Agr. Chem. Mon.) 16(6): 77-79; 1972.* (Japanese)

Although substances used for the control of ectoparasites on poultry legally fall under the veterinary drug regulations rather than the pesticide regulations in Japan, they often contain the same active ingredients as pesticide formulations and therefore represent a route for the occurrence of pesticide poisoning in poultry. A second route of pesticide ingestion by poultry is the ingestion of feed containing pesticides, either introduced intentionally in the course of production or resulting from accidental contamination. Studies by the U.S. Department of Agriculture revealed that organochlorine residues occur almost universally in poultry fat. After pesticide administration for a limited interval in laying hens, organochlorines continue to be excreted via the eggs for a considerable length of time. Organophosphate and carbamate insecticides, although having higher acute oral toxicities to birds than the organochlorines, are not stored appreciably in the fat and are reported to have no deleterious effects on eggshell formation. In addition, the chronic oral toxicities (daily administration of small quantities) of the organophosphates and carbamates in poultry are not very much greater than those of the organochlorines.

72-1895. Harrison, D. L. (Wallaceville Animal Res. Centre, Private Bag, Wellington, New Zealand). **Veterinary aspects of insecticides: organochlorines.** *N. Z. Vet. J. 19(10): 227-232; 1971.* (17 references)

Although organochlorines are not expected to be used in the future to any great extent in New Zealand, their use in the past has had a profound influence on economic pest control and experience gained with them is useful for other pesticides. Large-scale empirical attacks with insecticides seem to fail because of the resistance developed by the insects. Some of the more recently introduced compounds are showing the same trend in this regard as DDT. Sophisticated analytical methods developed for organochlorine determinations provide a background for residue studies of other chemicals. Work on the uptake and elimination of DDT and lindane has provided means of assessing from fat analysis how livestock were exposed to these chemicals. Residue patterns in humans have also been established after withdrawal of certain compounds. Environmental levels of DDT, the most widely used insecticide for 20 yr in New Zealand, appear, in general, to present little hazard to humans, domestic stock, or wildlife there.

72-1896. Solly, S. R. B. (Wallaceville Animal Res. Centre, Private Bag, Wellington, New Zealand). **Veterinary aspects of insecticides: organophosphates.** *N. Z. Vet. J. 19(10): 233-240; 1971.* (10 references)

Organophosphates produce toxicity to the nervous system by inhibition of AChE, an enzyme concentrated in the synaptic cleft of the cholinergic nerves. The normal dephosphorylation step is quite slow, and when the organophosphate combines with the enzyme, acetylcholine accumulates at the synapses, resulting in excessive stimulation, followed in severe poisoning by complete blockade. Defecation, urination, lachrymation, and slowing of the heart are initial signs of poisoning. One of the most specific and distinct symptoms is marked contraction of the pupils. Species differences in effects are noted. Twitching, paralysis, and sometimes convulsions are seen. Death is usually due to respiratory failure.

Erythrocyte ChE is useful as an indicator of exposure to organophosphates, although in the case of the more polar compounds it does not reflect the activity of the brain enzyme. Depot fat analysis is probably the most useful tissue assay for determination of the presence of organophosphates. Veterinary therapy includes the use of atropine to exclude acetylcholine from effector cells and 2-PAM to reactivate the enzyme. Flavones have been used experimentally to inhibit cholineacetylase [EC 2.3.1.6].

72-1897. Okada, H.; Kamata, T.; Nakagawa, A.; Hirakawa, M. (Author address not given). **[A case of acute poisoning caused by Gramoxone, an organochlorine [*sic*] pesticide (herbicide).]** *Nippon Kyobu-Geka Gakkai Zasshi (J. Jap. Ass. Thorac. Surg.) 10(Suppl): 55; 1972.* (Japanese)

A man who accidentally swallowed 15 ml of Gramoxone, an herbicidal formulation containing 26% paraquat, died on the 20th day post ingestion due to progressive dyspnea, despite recovery from acute nervous system and hepatic dysfunction. Extensive, severe pulmonary fibrosis was seen at autopsy. This was the second case of Gramoxone poisoning observed in the authors' hospital; in the first case, death occurred much sooner, due to the ingestion of a larger amount of the same formulation. The autopsy findings were correspondingly different.

72-1898. Ishikawa, T. (Kitazato U., Sch. of Med., Japan). **[Eye diseases caused by organophosphates.]** *Hokenfu Zasshi (Jap. J. Pub. Health Nurse) 27: 58-59; 1971.* (4 references) (Japanese)

The effects of the organophosphates (or carbamates) contained in parathion, EPN, DDVP (dichlorvos), diazinon, Sumithion (fenitrothion), malathion, Suncide (propoxur), Hopcide (CMPC), and especially their effects on the eyes are discussed briefly. Deterioration of vision, narrowing of vision, astigmatism, nervous system damage, deterioration of memory, excessive thirst, unbalanced appetite, occasional constipation or diarrhea, perspiration, loss of weight, viscosity in the mouth, staining of teeth, eye irritation, watering of the eyes, headaches, dizziness, pains in the shoulders, shortness of breath, palpitations, stomach and abdominal pains, and twitching of eyelids are common symptoms of organophosphate poisoning. As soon as these symptoms are noted, inquiries concerning the use of pesticides in the neighborhood and immediate consultation with a physician are advised. Administration of PAM (500 mg) in 5% glucose solution, padrin (3 to 6 tablets daily), thiora (thiola?) (3 tablets daily), or vitamin B12 is advised. Treatment for approximately 6 to 7 mo will cure most symptoms.

72-1899. Takagi, S. (Okayama U., Med. Sch., Dept. of Internal Med., Okayama, Japan). **[Studies on agricultural organofluoride poisoning. Part II. Influence of an organofluoride on electroencephalograms.]** *Okayama Igakkai Zasshi (J. Okayama Med. Soc.) 81(11/12): 633-640; 1971.* (Japanese)

Electroencephalograms (EEG) of five patients poisoned by an agricultural organofluoride, Nissol (MNFA), were studied. The patients were all male, between 21 and 35 years of age, and all were poisoned during or after employment in application of diluted Nissol sprays. One of the cases was considerably more serious than the other four, but there were no convulsive episodes such as those reported by Harrison and Brockmann. There was severe clouding of consciousness accompanied by a low level of blood glucose. The mild cases showed almost no abnormalities, but as the degree of poisoning increased, the EEG varied from low-voltage, irregular, slow waves to almost flat waves. Administration of glucose brought good responses in these mild cases. A close correlation was noted between impaired carbohydrate metabolism and hypoglycemia. This seems to be an important criterion in the differential diagnosis of organofluoride poisoning as compared to other types of pesticide poisoning. It was concluded that the EEG is a valuable adjunct in determining the severity of organofluoride poisoning.

72-1900. Chao, T. C. (Author address not given). **Death by organo-phosphorus poisoning in Singapore 1960-1969.** *Singapore Med. J. 12(2): 77-79; 1971.* (4 references)

Due to the availability of organophosphate insecticides, the percentage of deaths from these poisons in Singapore is increasing. Of the 43 cases reported between 1960 and 1969, 90% were suicides and 10% were accidents. Post-mortem examination revealed pulmonary edema and excessive bronchial secretion in almost all cases. No ulceration of the stomach was observed, but a milky white fluid with a kerosene or fruity smell was present when poison remained in the stomach. From 33 to 54 mg of organophosphate has been found in stomachs of those who died before gastric lavage was performed. In the 5 cases tested, 4 showed a serum AChE of 8 units; 1 of 16 units compared to a normal of 95-177 units; and all showed no activity at the neuro-muscular junction in the intercostal muscles.

72-1901. Chew, L. S.; Chee, K. T.; Yeo, J. M. S.; Jayaratnam, F. J. (Med. Unit III, Outram Road Gen. Hosp., Singapore). **Continuous atropine infusion in the management of organophosphorus insecticide poisoning.** *Singapore Med. J. 12(2): 80-85; 1971.* (7 references)

The major factor effecting recovery of patients who had intentionally ingested organophosphates was the use of a continuous infusion of atropine. Routinely, on admission, the patient's air passage was cleared, a

tracheostomy was performed, and anoxemia was corrected. Mild atropinization was achieved with i.v. injections and then continuous administration of 12 or 18 mg/pint were used to insure adequate atropinization regardless of other emergencies requiring ward personnel. Doses were adjusted to individual needs and reduced as clinical signs, not cholinesterase levels, improved. Two doses of 2-PAM were also administered, and antibiotics were given routinely. Stomach lavage was carried out after sufficient atropinization had been achieved. The only patient who did not survive despite this treatment, a 72-yr-old diabetic, succumbed to *Pseudomonas pyocyanea* septicemia from the tracheostomy.

See also **72-1833** **72-1926** **72-1930** **72-1966** **72-1969**

72-1902. Uzoukwu, M.; Sleight, S. D. (Dept. of Pathol., Michigan State U., East Lansing, MI 48823). **Dieldrin toxicosis: fetotoxicosis, tissue concentrations, and microscopic and ultrastructural changes in guinea pigs.** *Amer. J. Vet. Res. 33(3): 579-584; 1972.* (13 references)

Of 42 guinea pigs initially treated with dieldrin at 15 or 30 mg/kg body wt, and subsequently at 5-day intervals with 0 or 15 mg/kg, 30 died. No abortions occurred even though 30 pregnant females were used in the experiment. Fatty metamorphosis of hepatocytes and vacuolar degeneration of renal tubular epithelium were consistently observed upon histopathological examination. Lungs, but not brains, were also affected. Fetuses appeared normal on gross examination, and dieldrin was detected in fetal tissue from two groups of treated animals. Dieldrin at a concentration of 100 ppm in the feed or sodium nitrite at a concentration of 0.5% in the water induced abortion. Sodium nitrite did not seem to potentiate the abortion-inducing activity of dieldrin. Death occurred in one-third and one-half of the pregnant and nonpregnant females, respectively, at the 100 ppm level of dieldrin. In uteri of dieldrin- or nitrite-treated animals that aborted, focal epithelium vacuolation and necrosis were observed. Electron microscopic examination of brain tissue of male guinea pigs given dieldrin at 75 mg/kg body wt ip revealed swelling of the mitochondria and disintegration of associated internal cristae in the cerebral cortex and similar changes in the cerebellum.

72-1903. Martin, G. C.; Abdel-Gawad, H. A.; Weaver, R. J. (Dept. of Pomol., U. of California, Davis, CA). **The movement and fate of (2-chloroethyl)phosphonic acid in walnut.** *J. Amer. Soc. Hort. Sci. 97(1): 51-54; 1972.* (5 references)

^{14}C-Ethephon applied to a walnut leaflet was absorbed and translocated more rapidly in seedlings than in mature trees. Activity was low in the kernel and highest in the hulls regardless of method of application. Radioactivity in the kernel decreased rapidly between 5 and 7 days after application. TLC did not reveal any metabolites within the leaves, hulls, and kernels.

72-1904. Morita, R.; Lieberman, L. M.; Beierwaltes, W. H.; Conn, J. W.; Ansari, A. N.; Nishiyama, H. (Nuclear Med. and Endocrinol. and Metabolism Div., Dept. of Internal Med., U. of Michigan, Ann Arbor, MI 48104). **Percent uptake of ^{131}I radioactivity in the adrenal from radioiodinated cholesterol.** *J. Clin. Endocrinol. Metab. 34: 36-43; 1972.* (5 references)

The value of ^{131}I-19-iodocholesterol uptake by the adrenal gland as a technique for diagnosis of Cushing's syndrome was demonstrated. No overlap was observed between percent uptake of ^{131}I radioactivity in patients with normal adrenal function and those with Cushing's syndrome. The patient treated with *o,p'*-DDD at the time of administration of the radiocholesterol had the lowest percent uptake of ^{131}I in the Cushing's syndrome group, although it was still significantly greater than in normal patients.

72-1905. Skromme-Kadlubik, G.; Alvarez-Cervera, J.; Cortes-Marmolejo, F. (Comision Nacional de Energia Nuclear, Mexico City, Mexico). **Studies of suprarenal scintigraphy in humans using ^{131}I-DDD.** *J. Nucl. Med. 13(4): 282-284; 1972.* (10 references)

DDD (TDE) labeled with ^{131}I was tested as a diagnostic agent for tumors of the adrenal medulla or cortex. Scanning of 13 normal patients indicated that variations in concentration occurred between individuals and within an individual at different times of the day. In the five cases of suprarenal tumor studied, the size and location of the tumor could be shown by direct visualization. At the doses studied (200 to 350 μCi) no radiological toxicity was observed.

72-1906. Szeberenyi, J.; Varga, A.; Pongracz, M. F.; Molnar, J. (Dept. of Applied Toxicol., State Institute of Occupational Health, Budapest 9, POB 22, Hungary). **Centrally mediated contraction of the lower eyelid elicited by anticholinesterases in anaesthetized rats.** *J. Pharm. Pharmacol. 24(1): 85-86; 1972.* (9 references)

Recording the movements of the rat lower eyelid may provide an easy, reliable method for the study of central sympathetic hyperactivity caused by anticholinesterases. Anesthetized, immobilized, adrenalectomized rats, artificially ventilated, were monitored for blood pressure, intratracheal pressure, and movements of the lower eyelids after administration of several organophosphates. In each animal the cervical vago-sympathetic trunk was dissected and cut on one of the sides. Dichlorvos, at 2 to 4 mg/kg body wt, and mevinphos, at 200 to 400 μg/kg, elicited sustained contractions on the innervated but not on the decentralized lower eyelid, indicating an action of central origin. Peripherally acting drugs were active on both sides. The fact that a rise in intratracheal pressure was not observed in some cases had no causal connection with the contractions of the lower eyelid, since anticholinesterase-induced contractions were not prevented by blocking bronchoconstriction with a quarternary tropine derivative.

72-1907. Hrdina, P. D.; Singhal, R. L. (Dept. of Pharmacol., Faculty of Med., U. of Ottawa, Ottawa, Ont., Canada, K1N 6N5). **Neurotoxic effects of DDT: protection by cycloheximide.** *J. Pharm. Pharmacol. 24(2): 167-169; 1972.* (8 references)

Cycloheximide, injected in rats in a single dose of 70 μg/100 g 30 min before *p,p'*-DDT, prevented development of hyperpyrexia, tremors, and convulsions normally seen following administration of the same dose of DDT. In addition, the concentration of acetylcholine

in the striatum remained at control level, while in animals treated with DDT alone it was reduced by 38%, according to this Letter-to-the-Editor. Actinomycin D, administered in two doses totaling 40 μg/100 g 30 min before and 2.5 hr after DDT-treatment, did not affect the DDT-induced change in striatal acetylcholine or the development of neurotoxic signs; increase in body temperature was delayed and marked hyperpyrexia was seen only during the fifth hour after administration.

72-1908. Earnest, R. D.; Benville, Jr., P. E. (Bureau of Sport Fisheries and Wildlife, Div. of River Basin Studies, Sacramento, CA 95825). **Acute toxicity of four organochlorine insecticides to two species of surf perch.** *Calif. Fish Game 58(2): 127-132; 1972.* (14 references)

Static and intermittent-flow bioassay techniques were used to determine toxicity of endrin, DDT, aldrin, and dieldrin in shiner perch and dwarf perch. TL50 values as determined by intermittent-flow bioassay were lower than those determined by static bioassay and were considered to be more meaningful. Concentrations of less than 3 μg/l of the four compounds were found to be lethal to the perch, and endrin was the most toxic to both species. Residues in dead fish were generally lower than those found in survivors, although the ranges overlapped. Concentrations ranged from 0.07 mg/kg body wt for endrin in live fish to 2.33 mg/kg for dieldrin in dead fish. A considerable amount of dieldrin was found in aldrin-exposed fish, indicating fairly rapid metabolism.

72-1909. Ptashne, K. A.; Wolcott, R. M.; Neal, R. A. (Div. of Toxicol., Dept. of Biochem., Vanderbilt U. School of Med., Nashville, TN). **Oxygen-18 studies on the chemical mechanisms of the mixed function oxidase catalyzed desulfuration and dearylation reactions of parathion.** *J. Pharmacol. Exp. Ther. 179(2): 380-385; 1971.* (14 references)

Aerobic incubation of parathion with rabbit liver microsomes in a medium enriched with $H_2{}^{18}O$ (20 atom % excess) resulted in incorporation of approximately 18 atom % excess ^{18}O in the diethylphosphate and diethylphosphorothionate formed. The paraoxon formed contained no ^{18}O, as indicated by the lack of ^{18}O in diethylphosphate obtained when the paraoxon was hydrolyzed with rabbit serum. When $^{18}O_2$ was used as the atmosphere for incubation of parathion with rabbit liver microsomes, the diethylphosphate formed contained 37.3 atom % excess ^{18}O; none was found in the diethylphosphorothionate. The paraoxon formed was enriched with ^{18}O as indicated by the 42 atom % excess ^{18}O found in its hydrolysis product, diethylphosphate. These results suggest that the desulfuration and dearylation of parathion catalyzed by mixed function oxidase proceed via a cyclic intermediate formed by an enzyme-catalyzed direct oxygen atom addition at the phosphorus-sulfur double bond.

72-1910. Kacew, S.; Singhal, R. L.; Hrdina, P. D.; Ling, G. M. (Dept. of Pharmacol., Faculty of Med., U. of Ottawa, Ottawa, Canada). **Changes in kidney cortex gluconeogenic enzymes induced by 1,1,1-trichloro-2,2-bis-(*p*-chlorophenyl)ethane (DDT).** *J. Pharmacol. Exp. Ther. 181(2): 234-243; 1972.* (29 references)

Increased activities of the four key renal gluconeogenic enzymes were observed in rats as early as 1 hr after administration of DDT at 600 mg/kg body wt. Peak activity of up to 300% above normal occurred at 5 hr, and a dose response relationship was established at this time point. Levels of the four hepatic gluconeogenic enzymes tested at 5 hr were also significantly elevated by *p,p'*-DDT administration. Adrenalectomized animals showed a similar enzyme response to DDT treatment. Inhibitors of protein synthesis, administered before DDT, significantly reduced insecticide stimulation of the activities of pyruvate carboxylase, phosphoenolpyruvate carboxykinase, fructose-1,6-diphosphatase, and glucose-6-phosphatase from kidney. No sex differences were observed in the response to *o,p'*-DDT or *p,p'*-DDT. Chronic administration of 0.5 mg/100 g of DDT produced a small stimulation of both renal and hepatic gluconeogenic enzymes.

72-1911. Jefferies, D. J.; French, M. C. (The Nature Conservancy, Monks Wood Exp. Sta., Huntingdon, England). **Changes induced in the pigeon thyroid by *p,p'*-DDE and dieldrin.** *J. Wildl. Manage. 36(1): 24-30; 1972.* (23 references)

Homing pigeons were fed *p,p'*-DDE at 72, 36, and 18 mg/kg body wt or dieldrin at 4.2 and 1 mg/kg daily for 56 days. All birds given 72 mg/kg DDE died before the end of the experiment. A significant difference was observed between the mean weight (81.0 mg) of thyroids of *p,p'*-DDE-fed birds and the mean weight (44.8 mg) of controls. The mean thyroid weight (79.6 mg) of dieldrin-fed birds was also significantly higher than that of controls. Visual and computerized image-area estimation of thyroid activity showed considerable loss of colloid from the follicles associated with hyperplastic epithelia in birds fed either *p,p'*-DDE or dieldrin. Adrenal and liver weights seemed to decrease, but no significant changes in these parameters were observed following dieldrin administration.

72-1912. Hassall, K. A.; Forrest, T. J. (Dept. of Physiol. and Biochem., U. of Reading, England). **Reductive dechlorination of DDT by heated liver.** *Nature (London) New Biol. 236(68): 214-216; 1972.* (6 references)

DDT was rapidly converted anaerobically to DDD in a heated, 12,000 *g* supernatant of pigeon liver homogenate containing an NADPH-generating system and riboflavin. Conversion was inhibited by omission of the NADPH-generating system, riboflavin, or heating of the supernatant. Both NADPH and NADH could be

substituted for the NADPH-generating system. Carbon monoxide completely inhibited the conversion. It is suggested that heat-treated cells or cells killed in another way can release cofactors which may normally be bound to protein. Under anaerobic conditions in soil and lakes, riboflavin or other cofactors liberated during putrefaction may bring about conversion of environmental DDT to DDD (TDE).

72-1913. Schuphan, I.; Ballschmiter, K. (Institut fuer Anorganische Chemie und Kernchemie der Universitaet, D6500 Mainz, Germany). **Metabolism of polychlorinated norbornenes by *Clostridium butyricum*.** *Nature (London) 237(5350): 100-101; 1972.* (12 references)

Clostridium butyricum was found to metabolize hexachloro-norbornene, a moiety of the cyclodiene insecticides which according to the literature remains unchanged in the ecosystem. The three metabolites identified included tetrachloro-norbornene, a hydroxylated derivative, and tetrachloro-cyclodiene. Tetrachloro-norbornene and the hydroxylated derivative were found when pentachloro-norbornene was applied to the culture, and only the hydroxylated derivative could be detected when tetrachloro-norbornene was applied. No explanation could be given for the fact that tetrachloro-cyclodiene was found only when the starting material was hexachloro-norbornene. Yeast cultures and soil microorganisms, mostly actinomycetes, on agar plates failed to produce dechlorinated products of hexachloro-norbornene.

72-1914. Weaver, R. J.; Abdel-Gawad, H. A.; Martin, G. C. (Dept. of Viticulture and Enol., U. of California, Davis, CA 95616). **Translocation and persistence of 1,2-^{14}C-(2-chloroethyl)phosphonic acid (ethephon) in Thompson Seedless grapes.** *Physiol. Plant 26(1): 13-16; 1972.* (14 references)

One week after application of labeled ethephon to the surface of Thompson Seedless grapes, 62% of the initial radioactivity remained on the surface. Berries that had been injected with the compound showed no activity on the surface. No radioactivity was found on the skin surface or in methanolic extracts of the berries when ethephon was applied to the base of the upper surface of the first leaf above the cluster or to the peduncles. Radiochromatograms of berry extracts showed no labeled metabolites. Treatment of mature leaf near the base of a shoot resulted in movement of the ethephon to the tip, indicating a possible translocation in the phloem in a source-to-sink relationship.

72-1915. Bowes, G. W. (Scripps Institution of Oceanography, U. of California, San Diego, La Jolla, CA 92037). **Uptake and metabolism of 2,2-bis-(*p*-chlorophenyl)-1,1,1-trichloroethane (DDT) by marine phytoplankton and its effect on growth and chloroplast electron transport.** *Plant Physiol. 49(2): 172-176; 1972.* (31 references)

DDT at a concentration of 80 ppb did not affect the growth of seven marine phytoplankters studied, once cell division commenced. There was a 9-day lag period before normal growth commenced in *Skeletonema costatum*, even when inoculum was taken from cells growing in DDT medium during the logarithmic phase of growth. Morphological changes were not observed after DDT exposure. In most cases, more than 90% of the DDT was associated with the cells when analyzed near the end of the logarithmic phase of growth. The only metabolite observed was DDE; maximum conversion (7.4%) occurred with *D. tertiolecta*. Chromatograms of extracts from *C. huxleyi*, *A. carteri*, and *Porphyridium* sp. indicated no metabolites. Either DDT or DDE at a concentration of 20 μM inhibited electron transport in chloroplast particles of *D. tertiolecta* by about 50%. Addition of test compound after buffer and chloroplast suspension were added was important to prevent adsorption on the glass walls.

72-1916. Hiltibran, R. C. (Illinois Natural History Survey, Urbana, IL 61801). **A proposed biochemical mechanism of the toxic action of DDT.** *Trans. Ill. State Acad. Sci. 64(1): 46-54; 1971.* (45 references)

DDT at 5.9×10^{-4} g/ml and 5.9×10^{-6} g/ml inhibited oxygen uptake by bluegill liver mitochondria in the presence of succinate 100% and 40%, by respectively. No increase in inorganic phosphate was observed in the reaction medium. When α-ketoglutarate was used as substrate, inhibition was not as pronounced and no effect on phosphate uptake was seen. At 17.7×10^{-4} g/ml, DDT inhibited the hydrolysis of ATP in the presence of zinc and cadmium and increased the hydrolysis of ATP in the presence of magnesium. Literature is cited to support the suggestion that the primary effect of DDT is inhibition of electron flow from succinic acid to the cytochrome chain.

72-1917. Robertson, B.; Enhoerning, G.; Ivemark, B.; Malmqvist, E.; Modee, J. (Dept. of Pediat. Pathol., Karolinska Sjukhuset, Stockholm, Sweden). **Paraquat-induced derangement of pulmonary surfactant in the rat.** *Acta Paediat. Scand. Suppl. 206S: 37-39; 1970.* (3 references)

Female Sprague-Dawley rats were inoculated sc with a 2% aqueous paraquat solution at a concentration of 35 mg/kg body weight and sacrificed at intervals from 2 hr to 7 days later. Morphological observations were performed on some of the animals; in the others, alveolar washing was performed to provide fluid for evaluation of surface properties and determination of lipid content. The surface properties of the alveolar washings were markedly abnormal within 2 hr after paraquat injection and deteriorated progressively. The phospholipid content of washings from treated animals did

not differ significantly from controls. Symptoms of respiratory distress developed within 12-24 hr after treatment, became severe 2-3 days after injection, and began to subside after the 4th day. Focal atelectasis was morphologically observable 12 hr after paraquat injection; atelectasis reached a maximum 2-3 days after injection. Overdistension of bronchioles and alveolar ducts occurred within atelectatic areas. Increasing interstitial inflammation, intra-alveolar edema, focal intra-alveolar hemorrhage, and scattered hyaline membranes became evident following the 2d day after paraquat injection. Mural proliferation of cuboidal epithelium was prominent in many peripheral air spaces in animals killed 7 days after paraquat injection.

72-1918. Modee, J.; Ivemark, B. I.*; Robertson, B. (Dept. of Pediat. Pathol., Karolinska Sjukhuset, Stockholm, Sweden). **Ultrastructure of the alveolar wall in experimental paraquat poisoning.** *Acta Pathol. Microbiol. Scand. Sect. A 80: 54-60; 1972.* (27 references)

At various intervals after administration of 35 mg/kg body wt paraquat to rats, lungs were examined by electron microscopy. No ultrastructural lesions were noted at 4 hr, but at 12 and 18 hr irregular interstitial edema and engorgement of alveolar capillaries were observed. Twenty-four hours after paraquat injection these signs were more prominent, and lesions of the alveolar epithelium and the capillary endothelium were evident. The mitochondria of these tissues were swollen and apparently degenerating. By 48 hr there was widespread necrosis of the alveolar epithelium, observed in granular as well as membranous pneumocytes. Desquamated alveolar epithelial cells were found; many contained easily recognizable lamellar inclusions. The functional derangement of pulmonary surfactant seen in paraquat poisoning may be related to the severe ultrastructural lesions observed in the alveolar epithelium.

72-1919. Dikshith, T. S. S.; Datta, K. K. (Ind. Toxicol. Res. Centre, Chattar Manzil Palace, Lucknow, India). **Effect of intratesticular injection of lindane and endrin on the testes of rats.** *Acta Pharmacol. Toxicol. 31(1-2): 1-10; 1972.* (19 references)

Intratesticular injection of 0.25 mg of lindane in saline solution into 12 rats resulted in testicular atrophy in seven rats and testicular hypertrophy in five rats on the 10th day post injection. The hypertrophic testes were swollen to about twice normal size; histopathological observation revealed extensive degenerative changes in the seminiferous epithelium and intratubular and intertubular regions, as well as in the spermatogenic cells. Numerous multinucleated giant cells of the foreign-body type were present in the tubules and interstitial area. The atrophic testes were approximately one-third normal size; degeneration was characterized by shrunken tubules filled with edematous fluid, multinucleated giant cells, and inflammatory cells. The 0.25-mg endrin injection produced only slight hypertrophy under similar conditions. Upon histological examination, the tissues were found to be stained poorly. Spermatogenic cells were necrotic; the necrotic cellular elements of the seminiferous epithelium and tubules coalesced to form a mass. Giant cells were absent. No deaths occurred in the experimental groups, and the general condition of the rats appeared normal.

72-1920. Eto, M.; Sakata, M.; Sasayama, T. (Dept. of Agr. Chem., Kyushu U., Fukuoka, Japan). **Biological activities of *p*-ethylphenyl and *p*-acetylphenyl phosphates and their thiono analogs.** *Agr. Biol. Chem. 36(4): 645-650; 1972.* (14 references)

In a search for compounds having insecticidal but not neurotoxic properties, 10 phosphorothioate and 6 phosphate esters of *p*-ethyl- or *p*-acetylphenols were synthesized and examined for biological activity. All of the esters of *p*-acetylphenols inhibited human plasma ChE to a high degree, but *p*-ethylphenyl phosphates were comparatively inactive. Chemical modifications of *p*-acetylphenyl phosphates greatly affected their inhibition of human erythrocyte AChE. Diethyl-4-acetyl-3-methylphenyl phosphate was found to be a very specific inhibitor of housefly ChE. This compound had an LD50 of 0.50 μg/fly. *O,O*-dimethyl *O*-4-acetyl-3-methylphenyl phosphorothioate was observed to be most toxic in houseflies and least toxic in mice and did not cause ataxia in hens at doses of 1 g/kg body wt. In this series of compounds, as in a series related to saligen cyclic phosphate, bulky aryl esters were found to be neurotoxic and not insecticidal. Small alkyl esters were insecticidal but not neurotoxic. A metabolic study of *O,O*-diethyl *O*-*p*-ethylphenyl phosphorothioate in mice revealed no neutral metabolites. *p*-Ethylphenol, *p*-(α-hydroxyethyl)phenol and *p*-acetylphenol were detected using thin-layer chromatography.

72-1921. Soeda, Y.; Kosaka, S.; Noguchi, T. (Chem. Div., Nisso Inst. for Life Sci., Nippon Soda Co., Ltd., Oiso, Kanagawa, Japan). **Identification of alkyl 2-benzimidazolecarbamates as a major metabolite of thiophanates fungicide in/on the bean plant.** *Agr. Biol. Chem. 36(5): 817-823; 1972.* (5 references)

A major metabolite of thiophanate-methyl, isolated from the leaves of the bean plant after foliar treatment from a water culture medium of the plant and from plant homogenate, was identified as methyl 2-benzimidazolecarbamate. Elemental analysis, cochromatography with standard material, and IR, NMR, UV, and mass spectroscopy were used to identify the compound. Mass spectroscopy was also used to show that thiophanate-ethyl was fragmented to ethyl 2-benzimidazolecarbamate.

72-1922. Palmer, J. S.; Haufler, M.; Hunt, L. M.; Schlinke, J. C.; Gates, C. E. (Toxicol. Res. Lab., Vet. Sci. Res. Div., Agr. Res. Serv., U.S. Dept. of Agr., Kerrville, TX 78028). **Chronic toxicosis of sheep from the organic herbicide di-allate.** *Amer. J. Vet. Res. 33(3): 543-546; 1972.* (7 references)

A study of the effects of a gradual increase in daily exposure of sheep to di-allate indicated that initial toxicity occurred after about 180 g had been ingested. The 10 mg/kg dose did not result in cumulative toxic effects but appeared to produce a tolerance evidenced by delayed alopecia at higher doses. Body temperature-by-week interaction effects were highly significant for feed consumed, packed cell volume, erythrocyte count, and hemoglobin value; a sex-treatment interaction related to food consumption was also observed. Although carbamates are generally inhibitors of ChE, no significant decrease in ChE was observed.

72-1923. Wallnoefer, P.; Engelhardt, G. (Bayerische Landesanstalt fuer Bodenkultur, Pflanzenbau und Pflanzenschutz, Munich, Germany). **Der Abbau von Phenylamiden durch *Bacillus sphaericus*. [Degradation of phenylamides by *Bacillus sphaericus*.]** *Arch. Mikrobiol. 80: 315-323; 1971.* (17 references) (German)

Degradation of various phenylamide compounds used as herbicides and fungicides in agriculture was investigated by cultivating *Bacillus sphaericus* in flasks of minimal medium + yeast extract containing 50 μmol/l. of the respective compound. After 2-6 days the *B. sphaericus* culture filtrates were extracted with chloroform and examined for the presence of the original compounds by TLC and UV spectrophotometry, and for the presence of degradation products by gas chromatography. Aniline was detected in the extracts after incubation of the bacteria with 2-methylbenzoic acid anilide, 2-chlorobenzoic acid anilide, 2,5-dimethylfurancarboxylic acid anilide, pyracarbolid, carboxin, and propham. The extracts from monalide and monolinuron incubation yielded 4-chloroaniline; linuron yielded 3,4-dichloroaniline; metobromuron gave 4-bromoaniline; and maloran produced 3-chloro-4-bromaniline. Only oxycarboxin, monuron, and the metabolite 2-hydroxymethyl-5-methyl-furancarboxylic acid anilide appeared to undergo no degradation. In general, the acylanilides and the phenylcarbamate ester propham were broken down considerably faster than the *N'*-methoxyphenylureas. Detection of the free acids of some of the anilides in the extracts indicated cleavage at the amide bond.

72-1924. Roth, J. A.; Neal, R. A.* (Div. of Toxicol. Dept. of Biochem., Vanderbilt U. Sch. of Med., Nashville, TN 37203). **Spectral studies of the binding of *O,O*-diethyl *p*-nitrophenylphosphorothionate (parathion) to cytochrome P-450.** *Biochemistry 11(6): 955-960; 1972.* (11 references)

Results suggest that *O,O*-diethyl *p*-nitrophenylphosphate and *O,O*-diethylphosphorothionate, metabolic products of parathion in various animal species, are formed by a single mixed-function oxidase enzyme system with two binding sites for parathion on the terminal cytochrome P-450. Spectrophotometric studies with rabbit liver microsomes indicated that parathion was bound to cytochrome P-450. At low concentrations of parathion a shoulder in the type I spectra was present, and the absorption minimum was shifted to 414 nm. As the concentration increased, the shoulder disappeared and the absorption minimum shifted to 419 nm. The fact that a type II spectrum may have partially masked the type I spectrum at low concentrations was suggested since parathion inhibited the type II spectra of aniline. Using liver microsomes from normal and phenobarbital-treated rabbits, the type I spectra of parathion were partially inhibited by type I spectral compounds similar to hexobarbital. It was postulated that the part that remained unaffected by hexobarbital may be a binding site for parathion, which cannot bind hexobarbital. Parathion decreased the type I spectrum of hexobarbital to the same degree, 30% with normal microsomes and 50% with phenobarbital-induced microsomes. Complete inhibition of the type I spectra of parathion by benzphetamine was biphasic, also suggesting that parathion may bind to more than one type I site.

72-1925. Holland, P.; Parkes, D.; Shakespeare, J. (Medical Div., Chemical Defence Establishment, Porton Down, Wiltshire, England). **Concentrations of the oxime 2-hydroxyiminomethyl pyridinium methyl methane sulphonate (P2S) after intramuscular injection in humans.** *Brit. J. Pharmacol. 44(2): 368; 1972.* (7 references)

In 22 volunteers it was found that an im dose of 500 mg pralidoxime mesylate was sufficient to produce the plasma levels of 4.0 μg/ml necessary for maximal antidotal activity in organophosphate poisoning within 5 min. Twenty min after injection of 500 and 750 mg of the drug, plasma drug levels of 4.9 and 6.3 μg/ml, respectively, were obtained. Three hours after an oral dose of 4.0 g of pralidoxime mesylate, injection of 500 mg produced a peak plasma concentration of 14.8 μg/ml in 20 min. Given 6 hr after the oral dose, the peak value of the drug was 9.9 μg/ml. Three injections of 500 mg given at 20-min intervals 3 hr after the fourth oral dose gave a peak plasma level of 23.9 μg/ml. A dull ache persisting for 5 or 6 hr accompanied the 750-mg injections, with visual disturbances being experienced by more than half of the volunteers.

72-1926. Deichmann, W. B. (School of Med. Res. and Teaching Center of Toxicol., U. of Miami, P.O. Box 8216, Coral Gables, FL 33124). **Toxicology of DDT and related chlorinated hydrocarbon pesticides.** *J. Occup.*

Med. 14(4): 285-292; 1972. (11 references)

Results of studies undertaken over a period of 30 yr on DDT and related organochlorines are described. A study of DDT and its metabolites in brains and livers taken at autopsy of patients with histories of liver and neurological diseases revealed levels 2 to 2.5 times higher than those taken from people who died accidentally in the same geographical area. Levels were related to home and garden use of the pesticide. Lifetime studies in rats indicated that feeding aldrin, dieldrin, endrin, DDT, Aramite, methoxychlor, and thiourea resulted in an overall dose-related decrease in the incidence of tumors. Viability and lactation were the most sensitive indicators of adverse effects of organochlorines on reproduction in mice. In dogs, delayed estrus, reduction of libido, stillbirths, and lack of mammary development following a 14-mo DDT-aldrin feeding experiment indicated rather lasting metabolic changes. Aldrin and dieldrin fed to dogs along with DDT induced a much more pronounced retention of total DDT in fat. Severe starvation of rats resulted in marked loss of weight, marked loss of body lipids, and marked loss of DDT or aldrin from the tissue. Microsomal enzymes were, in general, stimulated. In females the concentration of pesticides in abdominal fat was increased. A review of the data available on DDT suggests that its effects on humans of different ages and both sexes be fully investigated. However, its usefulness dictates that it be evaluated fairly and not substituted by other pesticides about which even less is known.

72-1927. Naumann, K. (Deutsche Akademie der Landwirtschaftswissenschaften zu Berlin, Institut fuer Phytopathologie Aschersleben, DDR). **Zur Dynamik der Bodenmikroflora nach Anwendung von Pflanzenschutzmitteln. IX. Versuche mit den Herbiziden Kalkstickstoff, Simazin und Natrium-trichlorazetat. [Dynamics of the soil microflora following application of plant protection agents. IX. Experiments with the herbicides calcium cyanamide, simazine and sodium trichloroacetate.]** *Kuehn-Arch. 84(4): 329-350; 1970.* (91 references) (German)

Calcium cyanamide, applied to soil in field experiments at the rate of 6000 kg/ha resulted in a transient reduction in the total bacterial count, followed by stimulation. A maximum bacterial count was reached after 15 wk. An initial depression was not seen at an application level of 6 kg/ha. Simazine applied at 60 and 6 kg/ha produced a slight initial stimulation of bacterial growth, followed by normalization; sodium trichloroacetate at 1200 kg/ha failed to affect the total bacterial count. Counts of the individual physiological groups revealed effects parallel to the changes in the total bacterial count in the case of calcium cyanamide, except that the cellulose-decomposing bacteria showed disproportionately great stimulation. *Azotobacter* colonies in the soil-count plates showed a distinct reduction in frequently after cyanamide treatment. Simazine generally had a slight, positive effect on the growth of the various physiological groups, except during the second test year, a very dry year, when the protein-decomposing bacteria, nitrifying, cellulose-decomposing, and spore-forming bacteria were significantly reduced. Inhibition of protein-decomposing, anaerobic, and denitrifying bacteria, as well as soil algae, was seen during the dry test year after application of sodium trichloroacetate.

72-1928. Sacher, R. M. (Monsanto Co., 800 N. Lindbergh Blvd., St. Louis, MO 63166). **A mosquito larvicide with favorable environmental properties.** *Mosquito News 31(4): 513-516; 1971.* (6 references)

Exposure of mosquito larvae at each stage of development to 2,6-di-*tert*-butyl-4-(*α*,*α*-dimethylbenzyl) phenol resulted in normal larval development followed by blocked metamorphosis in the early stages of pupation and death. Late instars were as susceptible to the chemical as early instars, although there may have been a time during the fourth instar after which exposure did not result in death. The compound was extremely selective and had a low toxicity to fish and mammals. A minimum lethal dose of 10 to 50 ppm was established for eight species of fish. The oral LD50 in rats was found to be 1890 mg/kg, and the acute dermal MLD in rabbits was 3160 mg/kg. The half-life of 10 ppm of the insecticides in soil was about 2 days.

72-1929. Domschke, S.; Domschke, W.; Classen, M. (Medizinische Klinik Mit Poliklinik der Universitaet Erlangen-Nuernberg, Germany). **Zum Mechanismus der Leberzellschaedigung durch Alkylphosphate. [The mechanism of liver cell damage by alkyl phosphates.]** *Naturwissenschaften 58(11): 575; 1971.* (6 references) (German)

In an investigation of the mechanism of liver cell damage produced by alkyl phosphates, Wistar rats were injected sc with DFP at 1 mg/kg body wt or soman at 0.05 mg/kg. One hour or one day later, the rat livers were subjected to isolated perfusion. As an expression of cellular damage, the transaminases from the livers of treated rats were released into the perfusion medium more rapidly than those from normal livers. Liver samples were taken from treated animals by the freeze-stop technique. A distinct decrease in the ATP/ADP ratio was found, and consequently, a marked reduction in various energy-consuming synthesis processes. An appreciable shift in the redox equilibria of both mitochondrial and extramitochondrial compartments of the damaged cells was indicated by the elevated *β*-hydroxybutyrate/acetoacetate and lactate/pyruvate ratios. The observed lowering of cytosine nucleotide levels was attributable to reduced production of their precursor, uridine triphosphate. The observed effects of the alkyl phosphates were almost completely absent from animals given 4 mg/kg of atropine ip 15 min before alkyl phosphate injection. Thus liver damage was secondary and

could be ascribed to the generalized hypoxia resulting from cholinesterase inhibition. This hypoxia, along with the low consumption of reducing equivalents for biosynthetic processes, was responsible for the rise in the redox ratio.

72-1930. Wenzel, K.-D.; Dedek, W. (Forschungsstelle fuer Chemische Toxikologie der Deutschen Akademie der Wissenschaften zu Berlin, 705 Leipzig, DDR). **Isolierung und Identifizierung augenreizender Nebenprodukte der Dimethoat-Synthese. [Isolation and identification of eye-irritating byproducts of dimethoate synthesis.]** *Z. Chem. 11(12): 461-462; 1971.* (13 references) (German)

Severe eye irritation occurred in workers engaged in the industrial production of dimethoate during the neutralization of the acid intermediate $(MeO)_2PSSH$ in order to separate the process byproducts from the salt. Thin-layer chromatography produced four byproduct components, one of which was found, in rabbit eye irritation tests, to produce clouding of the cornea, unresponsiveness of the iris, severe reddening of the conjunctiva, and marked swelling of the eyelids. Attempts to concentrate this irritating component for study by solvent partitioning were unsuccessful; therefore, several possible compounds were synthesized for comparison. *Rf* values and color on TLC plates identical to those of the irritating component were given by the disulfide compound, bis(dimethoxythiophosphoryl) disulfide, which can presumably form during the commercial industrial synthesis of dimethoate by oxidation of the acid intermediate. A second rabbit eye irritation test revealed that the acid, $(MeO)_2PSSH$, is even more irritating to the eye than the disulfide compound, and acid formation in the eye by reduction of the disulfide compound is probably responsible for the observed irritation. This hypothesis was supported by the collection of ocular secretions from rabbit eyes at intervals after application of the radiolabeled disulfide, revealing appreciable acid formation within 2 hr after application.

72-1931. Ito, R.; Kawamura, H.; Toida, S.; Matsu-ura, S.; Sasaki, T.; Yanagisawa, M.; Chang, H. S. (Dept. of Pharmacol., Sch. of Med., Toho U., Omori, Tokyo, Japan). **Subacute toxicity of a new pesticide, *O,O*-dimethyl-*S*-isopropyl thioethyl phosphorodithioate (Z-7272) in mice and rats.** *Toho Igakkai Zasshi (J. Med. Soc. Toho Univ.) 19(1/2): 18-23; 1972.*

A new organophosphate insecticide, *O,O*-dimethyl-*S*-isopropyl thioethyl phosphorodithioate, was included in food of mice and rats at four dosage levels for 3 mo. No significant changes were observed in weight gain, food consumption, or blood or urine chemistry. A decrease of 20 to 30% in serum ChE was observed at the dosage of 12.5 ppm in females. No changes were found in brain ChE. Histopathological examination revealed interstitial small spotted cell-infiltrations in liver and kidney, but these were also found in some controls. One case of fat deposition in the adrenal medulla was found with the 12.5-ppm dose.

72-1932. Vij, S.; Kanagasuntheram, R. (Dept. of Anatomy, U. of Singapore, Fac. of Med., Sepoy Lines, Singapore). **Effect of tri-*o*-cresyl phosphate (TOCP) poisoning on sensory nerve terminations of slow loris.** ***(Nycticebus coucang coucang)*** *Acta Neuropathol. 20(2): 150-159; 1972.* (24 references)

Results of histochemical studies of sensory nerve terminations in the oral and digital tissues of slow loris confirmed the assumption that widespread degenerative change occurs in the peripheral sensory end-organs of tri-*o*-cresyl phosphate (TOCP)-poisoned animals. Following repeated topical application of a single oral dose of TOCP a reduced amount of AChE and a lack of ChE were found in sensory nerve terminations. Electron microscopy revealed vacuolization, degenerating mitochondria, distended endoplasmic reticulum, vesicle formation, and "cytosegregosomes" in the light cells of taste buds. Dark cells also had dense core granular vesicles of unusual shape. In the more peripheral nerve fibers, axoplasm was widely dispersed, faintly granular and contained few mitochondria or neurotubules and almost no agranular vesicles. There were fewer proximal deviations. Large numbers of "globular" masses along the nerve bundles in digital skin and in the hair end organs were seen by the Nauta-Gygax method.

72-1933. Lorber, M. (Dept. of Physiol. and Biophys., Georgetown U. Schools of Med. and Dent., Washington, DC). **Hematoxicity of synergized pyrethrin insecticides and related chemicals in intact, totally and subtotally splenectomized dogs.** *Acta Hepato-Gastroenterol. 19(1): 66-78; 1972.* (41 references)

Intact, and recently, chronically or partially splenectomized dogs were examined for hematotoxic effects following exposure on four consecutive days to fogs of some pesticides and related chemicals in a refined kerosene base. In intact and recently splenectomized dogs, 1-methyl naphthalene caused significant leukocytosis. Pyrethrins, 1-methyl naphthalene, butyl carbital and piperonal caused a slight, but significant reticulocyte elevation in chronically splenectomized dogs. Platelet and reticulocyte changes occurred less frequently and at a later time in intact animals than in splenectomized animals. Sequestering or metabolizing capabilities of the spleen may be responsible for the resistance of intact animals to hematotoxicity of pesticide fogs. However, the alteration of blood flow resulting from ligation of the splenic vein during surgery probably has the effect of prolonging the circulation of toxic compounds before their metabolism in the liver.

72-1934. Gasper, G. M.; Kawatski, J. A. (Dept. of Biol., Viterbo Coll., La Crosse, WI 54601). **Inhibition by heptachlor epoxide of succinic dehydrogenase activity in mouse liver homogenates.** *Comp. Biochem. Physiol. 41B: 655-660; 1972.* (4 references)

Spectrophotometric comparisons of the succinic dehydrogenase activity of mouse liver extracts were performed in the presence of several concentrations of heptachlor epoxide. Heptachlor epoxide had no effect on enzyme activity at concentrations of 0.166, 0.332, 1.66, and 3.32×10^{-5} *M*. At 16.6×10^{-5} *M* the insecticide caused slight inhibition of the enzyme system. (Author abstract by permission)

72-1935. Feldmann, R. J.; Maibach, H. I. (Dept. of Dermatol., U. of Calif., San Francisco, CA 94122). **Pesticide percutaneous penetration in man.** *J. Invest. Dermatol. 54: 435-436; 1970.*

Results of an investigation of the absorption of six representative pesticides through human skin indicate that this route represents a significant source for pesticide toxicity. Carbaryl had the highest maximum absorption rate (3% of dose/hr) and total percent absorbed as measured by appearance of administered radioactivity in the urine. Five days after application the total percentages of the pesticides absorbed were: 9.7 (parathion), 8.2 (malathion), 73.9 (carbaryl), 9.3 (lindane), 7.7 (dieldrin), and 7.6 (aldrin).

72-1936. Rickett, F. E.; Tyszkiewicz, K.; Brown, N. C. (Cooper Technical Bur., Berkhamsted, Herts., England). **Pyrethrum dermatitis. I. The allergenic properties of various extracts of pyrethrum flowers.** *Pestic. Sci. 3(1): 57-66; 1972.* (23 references)

Extracts of pyrethrum flowers were obtained by treatment with various aqueous and organic solvents. These were tested for immediate and delayed hypersensitivity reactions in guinea pigs previously sensitized to pyrethrum by single subcutaneous injection of the ground flowers suspended in Freund's complete adjuvant. Strong reactions were elicited by various aqueous extracts of the flowers; the allergens are of high molecular weight, being non-dialyzable. A hexane extract (similar to pyrethrum oleoresin) produced a moderate reaction, but a commercially refined pyrethrum extract proved negative. An extract of the spent flowers with methylene chloride showed irritant effects in unsensitized guinea pigs. (Author abstract by permission)

72-1937. Pope, G. G.; Ward, P. (E. African Community "Tropical Bird-Pest Res. Project," T.P.R.I., Arusha, Tanzania). **The effects of small applications of an organophosphorus poison, fenthion on the weaver-bird *Quelea quelea*.** *Pestic. Sci. 3(2): 197-205; 1972.* (6 references)

Experiments were carried out in East Africa to determine the effects of single-drop applications of the avicide Queletox (active ingredient fenthion) on the Red-billed Quelea *Q. quelea*, an important agricultural pest. Birds treated with relatively large amounts of Queletox died within a few hours, as expected. Most of those receiving the smallest lethal doses died 12 to 48 hr after treatment. From their terminal fat and muscle condition it was concluded that they died of starvation, despite their apparently healthy appearance following treatment and the easy availability of suitable food. LD50's of 80 and 220 mg/kg were obtained for Queletox applied to the feathers of the back in drops of varying sizes or concentrations, the amount of solvent having an important effect on the toxicity. The possibility is raised of reducing the amounts of Queletox used in control operations against the birds, making them safer and cheaper. (Author abstract by permission)

72-1938. Reid, W. D.; Christie, B.; Krishna, G.; Mitchell, J. R.; Moskowitz, J.; Brodie, B. B. (Lab. of Chem. Pharmacol., Natl. Heart and Lung Inst., NIH, Bethesda, MD 20014). **Bromobenzene metabolism and hepatic necrosis.** *Pharmacology 6: 41-55; 1971.* (15 references)

Periodic administration of 340 mg/kg piperonyl butoxide, which blocks hepatic microsomal enzymes, prevented liver damage in rats due to bromobenzene. Higher observed liver and plasma levels of bromobenzene indicated that piperonyl butoxide administration also blocked the metabolism of the hydrocarbon. These results, among with other findings, supported the conclusion that bromobenzene is normally metabolized to a hepatotoxic product, perhaps the epoxide.

72-1939. Sauter, E. A.; Steele, E. E. (Dept. of Anim. Ind., U. of Idaho, Moscow, ID 83843). **The effect of low level pesticide feeding on the fertility and hatchability of chicken eggs.** *Poultry Sci. 51(1): 71-76; 1972.* (8 references)

Ninety-six White Leghorn pullets housed in individual laying cages were used to study effects of low level pesticide in feed on fertility and hatchability of eggs in a 10-wk experiment. Pullets housed under similar conditions in a separate building served as controls. Hens were fed a commercial breeder ration. Feed samples were negative when analyzed for pesticides. DDT, diazinon, lindane, or malathion were added to the feed at rates of 0.1, 1.0 or 10.0 ppm. Commercially produced wettable powders were used to incorporate the insecticides in the feed. Hens were artificially inseminated twice each week using pooled semen from 10 males. Two test hatches at an interval of four weeks were incubated to determine levels of fertility and hatchability prior to pesticide feeding. Eggs produced were incubated beginning on the third day of pesticide feeding. Settings were made at weekly intervals with fertility determined on the seventh day of incubation. Pre-test fertility averaged 95% and hatchability 92% of fertile eggs. Fertility during the experimental period ranged from 96.1 to

99.0%. Hatchability of the control group during the experiment was 94.1%. All pesticides used except 0.1 ppm malathion significantly ($P < .05$) reduced hatchability. Embryonic mortality was increased throughout the incubation period. Egg shell thickness was significantly reduced by lindane and DDT. Egg production was reduced by all pesticides fed. (Author abstract by permission)

72-1940. Lillie, R. J.; Denton, C. A.; Cecil, H. C.; Bitman, J.; Fries, G. F. (U.S. Dept. of Agr., ARS, Anim. Sci. Res. Div., Beltsville, MD 20705). **Effect of *p,p'*-DDT, *o,p'*-DDT, and *p,p'*-DDE on the reproductive performance of caged White Leghorns.** *Poultry Sci. 51(1): 122-129; 1972.* (17 references)

Nine groups of 20 caged White Leghorn pullets each were fed a standard breeder diet supplemented with 5, 25, or 50 ppm *p,p'*-DDT, *o,p'*-DDT, or *p,p'*-DDE in experiment 1 (28 weeks); the 5 and 25 ppm levels were increased to 150 and 300 ppm, respectively, in experiment 2 (additional 12 weeks). A tenth group fed no pesticides served as the control in both experiments. Least squares analysis of variance indicated in experiment 1 pesticide effects for body weight gains which showed a rank of decreasing order: *p,p'*-DDT > *o,p'*-DDT > *p,p'*-DDE. The three pesticides produced significantly heavier body weights than the control. The level X pesticide effects indicated that the 5 and 25 ppm levels of *p,p'*-DDE increased body weight gains more than the 50 ppm level and that the 5 and 50 ppm levels of *p,p'*-DDT and *o,p'*-DDT, respectively, significantly reduced hatchability as compared with the other two levels within the pesticide. In experiment 2, pesticide effects showed that the three pesticides significantly reduced egg production; no differences were noted between the pesticides for this trait. No significant differences due to pesticides, as compared with the control, were observed for mortality, fertility, hatchability and progeny growth in both experiments. (Author abstract by permission)

72-1941. Cecil, H. C.; Fries, G. F.; Bitman, J.; Harris, S. J.; Lillie, R. J.; Denton, C. A. (U. S. Dept. of Agr., ARS, Anim. Sci. Res. Div., Beltsville, MD 20705). **Dietary *p,p'*-DDT, *o,p'*-DDT, or *p,p'*-DDE and changes in egg shell characteristics and pesticide accumulation in egg contents and body fat of caged White Leghorns.** *Poultry Sci. 51(1): 130-138; 1972.* (35 references)

White Leghorn pullets were fed diets containing 5, 25, and 50 ppm of *p,p'*-DDT, *o,p'*-DDT, or *p,p'*-DDE for 28 wk. The pesticides were then fed at a level of 50, 150, and 300 ppm for the succeeding period of 12 wk. Egg weight, egg shell thickness, and egg shell calcium were unaffected by treatment. Pesticide concentration of the egg contents reached equilibrium at 12 wk, and was approximately equal to dietary level for the *p,p'*-DDE and *p,p'*-DDT diets. The level of *o,p'*-DDT in eggs, however, was only 10% of the dietary level. At 2 wk 87% of the pesticide egg residue in *p,p'*-DDT-fed chickens was *p,p'*-DDT while 13% was *p,p'*-DDE. The proportion of *p,p'*-DDE increased until 12 wk when 25% of the pesticide residue was *p,p'*-DDE and remained at this level thereafter. There was no evidence for conversion of *o,p'*-DDT to *p,p'*-DDT in the chickens fed *o,p'*-DDT. At the end of the experiment, residues in the fat from birds fed *o,p'*-DDT equaled the concentration in the diet. However, birds fed *p,p'*-DDT or *p,p'*-DDE had residues in the fat which were 13-14 X that of the diet. Pesticides in the fat were approximately 13 X that of residues in egg contents. (Author abstract by permission)

72-1942. Richert, E. P.; Prahlad, K. V. (Dept. of Biol. Sci., Northern Illinois U., DeKalb, IL 60115). **Effects of DDT and its metabolites on thyroid of the Japanese quail, *Coturnix coturnix japonica*.** *Poultry Sci. 51(1): 196-200; 1972.* (10 references)

Histological and ^{125}I uptake studies of the thyroid gland of Japanese quail were conducted on birds that had been fed food treated with chlorinated pesticides for 120 days, and then returned to normal chow for 85 days. The pesticides and dosages used were as follows: control, 200 ppm DDA, 100 ppm DDT, and 150 ppm DDE. The histological picture of some of the experimental birds was similar to that displayed in colloid goiter, with a decrease in follicular resorption vacuoles and an increase in follicular size. For the 150-ppm DDE fed group, the absolute weight of the thyroid glands increased significantly, and the ^{125}I uptake decreased significantly. Apparently, even 85 days after chlorinated pesticide treatment was discontinued, the 150-ppm DDE fed Japanese quail exhibited a hypothyroid condition. (Author abstract by permission)

72-1943. Muller, H. D.; Lockman, D. C. (Dept. of Avian Sci., Colorado St. U., Ft. Collins, CO 80521). **Fecundity and progeny growth following subacute insecticide ingestion by the Mallard.** *Poultry Sci. 51(1): 239-241; 1972.* (9 references)

The influences of subtoxic levels of dieldrin and parathion on the fecundity and progeny development of the Mallard were investigated. Five trios each of mallards received either 4.0 ppm of dieldrin or 10.0 ppm of parathion in their diets; six additional trios constituted controls. The diets were fed *ad libitum* for 90 days, which included approximately 30 days prior to and 60 days following the onset of egg production. Egg production, fertility, hatchability, and progeny development to six weeks of age were evaluated. No significant influences on either egg production or fertility were disclosed. Hatchability of fertile eggs from dieldrin fed trios was significantly depressed to less than 50 percent of control or parathion levels. Egg shell thickness was significantly depressed in eggs from parathion treated individuals. A non-significant depression of shell thickness was also

noted in dieldrin shells. Shell thinning *per se* apparently was not responsible for the reduced hatchability of dieldrin embryos. Progeny from treated and control groups demonstrated equivalent hatching weights and rates of growth to six weeks of age. (Author abstract by permission)

72-1944. Sherman, M.; Herrick, R. B. (Coll. of Trop. Agr., U. of Hawaii, Honolulu, HI 96822). **Chronic toxicity and fly control from feeding S 4087 (*O*-*p*-cyanophenyl *O*-ethyl phenylphosphonothioate) to laying hens.** *Poultry Sci. 51(3): 1064-1066; 1972.* (6 references)

Technical grade S 4087 (*O*-*p*-cyanophenyl *O*-ethyl phenylphosphonothioate) was administered continuously at 100 ppm in the feed of White Leghorn laying hens for 40 wk. The droppings from these hens were moderately to highly toxic to maggots of *Parasarcophaga argyrostoma* (Robineau-Desvoidy), *Fannia pusio* (Wiedemann), *Chrysomya megacephala* (F.), and *Musca domestica* (L.). Treatment had no significant effect on egg weight, egg quality, egg flavor, and egg odor. On the other hand, treatment had a slight inhibitory effect on blood plasma cholinesterase activity, caused considerable hen mortality, and significantly depressed body weight, feed consumption, and egg production. Therefore, despite the good fly control obtained, S 4087 cannot be administered orally to laying hens to protect against the breeding of maggots in the droppings from these hens. (Author abstract by permission)

72-1945. Hunn, J. B. (Bur. of Sport Fisheries and Wildlife, Fish Control Lab., La Crosse, WI 54601). **The effects of exposure to Thanite on the blood chemistry of carp.** *Progr. Fish Cult. 34(2): 81-84; 1972.* (12 references)

The blood chemistry of carp was examined during exposure to 3 ppm Thanite (isobornyl thiocyanoacetate). During a 12-hr exposure, blood lactic acid increased more than 20 times and carbon dioxide decreased by a factor of 4. A decrease in blood pH was observed. Inorganic phosphate levels were 3 times those of controls. The fish, found lying on the bottom of the tank, recovered after 10 hr in fresh water and all parameters examined except for glucose, total protein, inorganic phosphate and lactic acid returned to normal in this time. The hypothesis of cyanide poisoning seems to present the best explanation for the results.

72-1946. Yudina, T. V.; Novikov, Yu. V. (F. F. Erisman Res. Inst. of Hyg., Moscow, USSR). **Izmeneniye pronitsayemosti gisto-gematicheskikh bar'yerov pod vliyaniyem khimicheskikh faktorov vneshney sredy. [Changes in the permeability of blood-tissue barriers occurring under the action of environmental chemical agents.]** *Gig. Sanit. 36(11): 14-18; 1971.* (13 references) (Russian)

Chlorophos (trichlorfon) and zineb, ^{35}S-labeled, were administered to white rats at 1/5000, 1/500, and 1/50 LD50 to study their effects on the blood-brain barrier and blood-endocrine organ barriers. The kidney was the most permeable organ while the brain was the least permeable. The permeability of the pituitary and the testes was inversely proportional to the dose of trichlorfon. The decrease in permeability of the spleen, kidney, adrenals, pancreas, thyroid, and liver was directly proportional to the concentration of zineb. Combined action of the two compounds resulted in a dose-dependent blood permeability decrease in all organs studied. The decrease in blood-barrier permeability caused by trichlorfon and zineb was associated with the hormonal impairment caused by these compounds.

72-1947. McBain, J. B.; Yamamoto, I.; Casida, J. E. (Div. of Entomol., U. of California, Berkeley, CA 94720). **Mechanism of activation and deactivation of Dyfonate (*O*-ethyl *S*-phenyl ethylphosphonodithioate) by rat liver microsomes.** *Life Sci. (Pt. II) 10(16): 947-954; 1971.* (11 references)

In the rat liver microsome-reduced nicotinamide-adenine dinucleotide phosphate system, the thionosulphur of Dyfonate (*O*-ethyl *S*-phenyl ethylphosphonodithioate) is replaced by oxygen from atmospheric molecular oxygen, producing the oxon analog, a potent anticholinesterase; however, in the same system, cleavage of the ester bond also occurs to form a non-toxic thiophosphonic acid derivative, incorporating oxygen from water, only. There is evidence that these reactions involve a common intermediate, a mechanism probably applicable to other thionophosphorus insecticide chemicals. (Author abstract by permission)

72-1948. Engst, R. (Zentralinst. fuer Ernaehrung in Potsdam-Rehbruecke der Deut. Akad. der Wissenschaften zu Berlin, DDR). **Zum Metabolismus ausgewaehlter Pflanzenschutz- und Schaedlingsbekaempfungsmittel. [Metabolism of selected plant-protection agents and pesticides.]** *Nahrung 15(8): 815-825; 1971.* (32 references) (German)

To demonstrate the occurrence and significance of pesticide conversion products in relation to the residue question, recent findings on pesticide metabolism are reviewed. In most organisms, the metabolism of DDT leads to the formation of water-soluble products which are readily excreted, and thus represents appreciable detoxification. In warm-blooded animals, this conversion seems to proceed only until a certain equilibrium level is reached. In the case of the organophosphates, one of the initial degradation steps (oxidation) leads to an increase in acute toxicity over that of the original compound. The *in vitro* degradation of the dithiocarbamate fungi-

cides leads to a variety of products with different toxicities, including some with possible carcinogenic, mutagenic, and teratogenic effects. The metabolism of the fungicidal phthalimides entails reaction with vital amino acids, and therefore the toxicity of these fungicides is appreciably higher in protein-deficient organisms than in those supplied with adequate protein. This finding is particularly important in relation to the use of these products in the developing nations, where dietary deficiencies are common. Continued research in pesticide metabolism and residue analysis is vital.

72-1949. Smith, J. N. (Dept. of Biochem., Victoria U. of Wellington, Wellington, New Zealand). **Detoxication mechanisms.** *N. Z. Vet. J. 19(10): 241-245; 1971.* (5 references)

Mechanisms of detoxication in animals are discussed in relation to insecticides. Metabolism of these compounds often involves several steps and can lead to production of either more or less toxic intermediates. Conjugation is one the major reactions for handling foreign compounds, but insecticides in general lack the functional groups necessary for combination with naturally occurring compounds. Introduction of the those functional groups is often the key mechanism by which insecticides are altered. Many instances of specificity are due to minor differences in activation and deactivation between insects and vertebrates. Synergistic compounds often alter the level of enzymes important in the activation or deactivation process.

72-1950. Sergeant, M.; Blazek, D.; Elder, J. H.; Lembi, C. A.; Morre, D. J. (Dept. of Botany and Plant Pathol., Purdue U., Lafayette, IN 47907). **The toxicity of 2,4-D and picloram herbicides to fish.** *Proc. Indiana Acad. Sci. 80: 114-123; 1970.* (24 references)

Low toxicity was observed in an investigation of the effects of picloram and 2,4-D on the green sunfish (*Lepomis cyanellus*). At $5 \times 10^{-4} M$, neither the acid nor the commercial salt formulations of 2,4-D produced toxic signs, but the butoxyethanol ester was toxic after 60 min. of exposure. The 99% analytical grade picloram was not toxic at $5 \times 10^{-4} M$, but the technical material and the 22% commercial formulation quickly immobilized the fish, suggesting the presence of a toxic impurity. Fish recovered in water without herbicide at a faster rate following repeated exposure. Ultrastructural changes were observed in the liver at a concentration of $10^{-4} M$ technical picloram. Longer periods of exposure did not result in more pronounced changes. One of the impurities tested, 2-(3,4,5,6-tetrachloro-2-pyridyl) guanidine, was toxic.

72-1951. Lichtenstein, E. P.; Fuhremann, T. W. (Dept. of Entomol., U. of Wisconsin, Madison, WI 53706). **Activity of an NADPH-dependent nitroreductase in houseflies.** *Science 172(3983): 589-591; 1971.* (8 references)

A nitroreductase, reducing parathion to aminoparathion, was found in the soluble fraction that was obtained from abdomens of female houseflies. The reaction required reduced nicotinamide adenine dinucleotide phosphate (NADPH), but was not affected by the presence or absence of oxygen. Further degradation of aminoparathion into water-soluble compounds occurred in NADPH-fortified incubation mixtures over prolonged incubation periods. The effect of sesamex or SKF 525-A on these reactions is described. (Author abstract by permission. Copyright 1971 by the American Association for the Advancement of Science.)

72-1952. Jackson, W. B.; Kaukeinen, D. (Environ. Studies Center, Bowling Green State U., Bowling Green, OH 43403). **Resistance of wild Norway rats in North Carolina to warfarin rodenticide.** *Science 176(4041): 1343-1344; 1972.* (7 references)

Reports concerning the ineffectiveness of warfarin rodenticide, used at farms and stores in a rural area about 8 kilometers in diameter near Raleigh, North Carolina, prompted subsequent laboratory testing. All of the 25 Norway rats trapped from the area survived 6 days of no-choice feeding of bait containing 0.025 percent (by weight) warfarin. (Author abstract by permission. Copyright 1972 by the American Association for the Advancement of Science.)

72-1953. Brankov, K. (Instituta za Farmakologiju i Toksikologiju, Medicinskog Fakulteta, Sarajevo, Yugoslavia). **Efikasnost protuotrova kod intoksikacije somanom u zavisnosti od vremena aplikacije. [The efficacy of antidotes in intoxication with soman depending on the time of application.]** *Veterinaria (Sarajevo) 20(2): 235-245; 1971.* (Croatian)

The *in vitro* antagonism of the effects of soman and its quaternary and tertiary thiocholine analogs by the antidotes atropine, benactisine, TMB-4, PAM-2, and MINA was investigated. The isolated rat bladder was used as the investigational model, representing the cholinergic nerve-transmitter-unstriated muscle system. The possibility for protection against poisoning is limited because of the rapid, irreversible inactivation of cholinesterase and the aging of the soman-cholinesterase complex, as well as steric hindrance. The effect of the reactivators and cholinolytics used in these experiments is to cause the relaxation of the bladder detrusors, and thus the combined use of these reactivators and cholinolytics has a therapeutic effect on the soman-poisoned rat bladder *in vitro*. Oximes and cholinolytics bring about complete functional recovery if given, at the latest, 7.5 minutes after poisoning with soman and its analogs. In the isolated rat bladder, the soman-cholinesterase reaction results in the complete aging of the enzyme within 120 min; however, complete aging does not occur within this

time period in the case of the thiocholine analogs. (From author abstract)

72-1954. Kudzina, G. D.; Golovan', D. I. (A. N. Marzeyev Res. Inst. for Gen. and Communal Hyg., Kiev, USSR). **Sravnitel'naya otsenka gerbitsidov gruppy khlorbenzoynoy kisloty sanitarno-toksikologicheskim metodom i metodom kul'tur kletok. [Comparative sanitary-toxicological and cell culture evaluation of the chlorobenzoic acid herbicide group.]** *Vrach. Delo 1: 125-128; 1972.* (Russian)

Parallel tests with several cell cultures and chronic toxicity studies were performed by po administration of dianate (2-methoxy-3,6-dichlorobenzoic acid, dicamba) and 2,3,6-trichlorobenzoic acid (2,3,6-TBA) to rats and rabbits. Dicamba was given in 7.5, 0.75, and 0.075 mg/kg doses and 2,3,6-TBA was administered in doses of 5.0, 0.50, and 0.05 mg/kg daily for 6 mo. The oxidoreductase enzymes in the rabbit were most susceptible to both compounds. Rabbit blood catalase, peroxidase, and serum oxidase activities were stimulated considerably. 2,3,6-TBA altered carbohydrate metabolism and caused minor shifts in CNS activity. Dicamba was toxic only at the 7.5 mg/kg dose while 2,3,6-TBA was toxic at 0.5 mg/kg. *In vitro*, trypsinized human embryo fibroblasts and monkey kidney cells were most susceptible to 2,3,6-TBA, producing dose-dependent cell degeneration from 150-750 mg/l. Further studies are recommended in the utilization of cell cultures for a rapid hygienic evaluation in the handling of organochlorine herbicides.

72-1955. Rodgers, C. A.; Stalling, D. L. (Fish-Pestic. Res. Lab., Bur. of Sport Fish. and Wildlife, Columbia, MO 65201). **Dynamics of an ester of 2,4-D in organs of three fish species.** *Weed Sci. 20(1): 101-105; 1972.* (9 references)

The uptake of ^{14}C-labeled butoxyethanol ester of (2,4,-dichlorophenoxy)acetic acid (BEE of 2,4-D) from water by three species of fed and fasted fish was studied. Fish were exposed to either 0.3 or 1.0 mg/l. of herbicide for up to 168 hr. Combined residues of the 2,4-D ester and its metabolic products were determined radiometrically in eight tissues and organs. Extracts of these organs were examined by thin-layer chromatography and autoradiography. Maximum residue concentrations were observed in most organs of fed fish within 1 to 2 hr of exposure, and within 1 to 8 hr of exposure for fasted fish. After maximum residue concentrations were reached, the herbicide and/or its metabolites were eliminated rapidly. (Author abstract by permission)

72-1956. Thompson, Jr., L. (Dept. of Agron., U. of Kentucky, Lexington, KY 40506). **Metabolism of simazine and atrazine by wild cane.** *Weed Sci. 20(2): 153-155; 1972.* (16 references)

Wild cane (*Sorghum bicolor* (L.) Moench) absorbed equal amounts of 2-chloro-4-(ethylamino)-6-(isopropylamino)-*s*-triazine (atrazine) and 2-chloro-4,6-*bis*(ethylamino)-*s*-triazine (simazine). The plants metabolized 70% of the atrazine and 30% of the simazine absorbed and translocated to the shoot during 24 hr. The major metabolites formed were hydroxy-derivatives and very hydrophilic metabolites which were chromatographically identical to peptide conjugates of atrazine, e.g., *S*-(4-ethylamino-6-isopropylamino-*s*-triazinyl-2)-glutathione and *S*-γ-L-glutamyl-(4-ethylamino-6-isopropylamino-*s*-triazinyl-2)-L-cysteine. Wild cane formed peptide conjugates of atrazine more rapidly than of simazine, but hydroxylation of these *s*-triazines occurred at approximately the same rate. (Author abstract by permission)

72-1957. Kuliyeva, A. M.; Rozengart, V. I.; Shmeleva, V. G. (Dept. of Biochem., I. P. Pavlov First Med. Inst., Leningrad, USSR). **Deystviye dvukh ryadov fosfororganicheskikh ingibitorov na kholinesterazu zritel'nogo gangliya kal'mara. [The effects of two series of organophosphate-type inhibitors on cholinesterase from squid optical ganglion.]** *Zdravookhr. Turkm. 14(7): 38-40; 1970.* (Russian)

A lyophilized cholinesterase preparation isolated from squid optic ganglia was employed in studying the structure of the hydrophobic region at the active site of cholinesterase. The susceptibility of this enzyme to two homologous series of organophosphates was determined. One of these series (I) comprised *O*-alkyl-*S*-butylmethylthiophosphates with alkyl chain lengths between 1 and 10, and the other (II), *O*-alkyl-*S*-hexylmethylthiophosphates with alkyl chain lengths between 1 and 7. Anticholinesterase activity was evaluated according to the bimolecular inhibition rate constant, computed via the pseudo-monomolecular reaction formula. Maximal enzyme inhibition was produced by the heptyl derivative of series (I) and by the butyl derivative of series (II). The active region of the esteratic site of the enzyme apparently includes two hydrophobic segments, on which sorption of the alkoxy group of the inhibitor takes place. One of these segments, corresponding to the butyl-containing alternative of series (II), is apparently followed by a short hydrophilic region, which is then linked to the other hydrophobic segment. The total length of the two hydrophobic segments including the hydrophilic portion is complementary to the heptyl alkoxy group of the series (I) inhibitors.

72-1958. Dahm, P. A. (Dept. of Entomol. and Zool., Iowa State U., Ames, IA). **Chemistry and metabolism of insecticides.** *In: Agricultural Practices and Water Quality, T. L. Willrich and G. E. Smith, ed., Iowa State Univ. Press, Ames, Iowa, 1970, pp. 167-182.* (62 references)

The metabolism of three major classes of insecticides is discussed, using the eight insecticides employed

most in the United States in 1964 as examples. DDT is metabolized by a number of routes, the best known of which is dehydrochlorination to DDE, the mechanism present in insects. Recent studies on dieldrin, aldrin, and heptachlor revealed hydroxylation, elimination of chlorine atoms and hydrolysis of epoxide rings in these chemicals, which were previously believed to be epoxidized and excreted in the feces. The metabolism of toxaphene has not yet been elucidated and therefore its fate in animals and natural waters is unknown. Parathion is converted to the highly toxic paraoxon by desulfuration *in vivo*. All of the other metabolites of parathion are less toxic than the parent compound. Malathion undergoes a similar desulfuration, but further detoxification mechanisms are responsible for its low mammalian toxicity. Carbaryl is readily metabolized by mechanisms which include hydroxylation at the *N*-methyl group and at two locations on the napthyl ring and epoxidation followed by epoxide cleavage and hydrolysis on the nonphenolic ring. In mammals the products are excreted as sulfates or glucuronides.

72-1959. Chu, J. P.; Kirsch, E. J. (Environ. Eng. Dept., Sch. of Civil Eng., Purdue U., West Lafayette, IN 47907). **Metabolism of pentachlorophenol by an axenic bacterial culture.** *Appl. Microbiol. 23(5): 1033-1035; 1972.* (3 references)

In conjunction with efforts to determine the feasibility of biological treatment of industrial wastewater containing PCP and other phenolics, a bacterium was isolated from a continuous-flow enrichment culture containing a final concentration of 200 mg/l. pentachlorophenol (PCP) and tested for its ability to metabolize PCP in a basal salts medium. In 84 hr, the PCP concentration in a test culture was reduced 80% and the viable cell count increased 50%. In cultures without PCP, neither a reduction in PCP nor an increase in cell count was observed. Using randomly labeled ^{14}C-PCP, 73% of the ^{14}C-PCP added appeared as respired $^{14}CO_2$ and no detectable residual PCP was found in the culture medium after 24 hr.

72-1960. Ishiguro, T.; Saito, T.; Toyoda, I. (Aburahi Labs., Shionogi & Co., Shiga, Japan). **[Fundamental research on the application of systemic insecticides. III. Fate of vamidothion in rice plant and citrus plant.]** *Bochu-Kagaku (Sci. Pest Contr.) 36(4): 159-168; 1971.* (16 references) (Japanese)

Using the ^{14}C-labelled product, the residue, persistence, and metabolism of vamidothion were studied in foliar application to rice and citrus plants. The highest residues of chloroform-extractable radioactive materials in polished rice were obtained when labeled vamidothion was applied in the flowering stage of the plant. Radioactive residues in chloroform extracts of rice bran were 10 times higher than in polished rice. When application was made later than in the flowering stage, larger quantities of radioactivity appeared in the husk than in the grain. Residues in the rice leaf 14 days after application averaged 0.17 ppm and 0.28 ppm for two different rice varieties. Degradation of vamidothion in citrus leaves increased with increasing temperature. Paper chromatography of the chloroform-extractable vamidothion metabolites in citrus leaves 8 days after application revealed the presence of vamidothion, vamidothion sulfoxide, and vamidothion sulfone in leaves kept at reduced temperatures. Vamidothion sulfoxide was found to be the principal metabolite of vamidothion in citrus leaves.

72-1961. Miyamoto, J. (Res. Dept., Pesticide Div., Sumitomo Chemical Co., Ltd., Osaka, Japan). **[Organophosphorus insecticides and environment (continued).]** *Bochu-Kagaku (Sci. Pest. Contr.) 36(4): 189-219; 1971.* (361 references) (Japanese)

This is a review article covering extensively the behavior of organophosphates in plants. Topics covered are organophosphate metabolism, distribution/storage, absorption, excretion, biotransformation, residues in food, decomposition and removal during food processing, residues and degradation in soil, and the effects of plant residues on wildlife, especially on birds and fish. Chemical formulas showing metabolism of some of the organophosphates in plants are illustrated. By and large, organophosphates have a strong tendency to degrade and disappear during food processing and cooking, and relatively small amounts of residues are found in processed food compared to the amounts in fresh vegetables and fruits.

72-1962. Patil, K. C.; Matsumura, F.; Boush, G. M. (Dept. of Entomol., U. of Wisconsin, Madison, WI 53706). **Metabolic transformation of DDT, dieldrin, aldrin, and endrin by marine organisms.** *Environ. Sci. Technol. 6(7): 629-632; 1972.* (16 references)

The metabolism of DDT and other chlorinated hydrocarbons was investigated under oceanic conditions and in pure microbial cultures. Oceanic conditions were simulated by incubating 1000-ml water samples with 0.1 μM of radiolabeled insecticide and at 23°C for 30 days. Of 100 microbial isolates tested, 35 seemed active in degradation of DDT, with TDE being the predominant metabolite. Degradation studies were performed using laboratory colonies of *Donaliella* sp., and *Agmenellum quadraplicatum* strain PF-6; field-collected, algae-containing water samples; surface film samples; and sediment samples, exposed to ^{14}C-DDT for 30 days at 30°C without illumination. All samples showed low metabolic activity toward DDT, the major metabolite being TDE, with some DDNS and DDOH. These samples were also incubated with dieldrin, aldrin, and endrin for one month. These insecticides were not metabolized in the open sea water or the estuarine samples, but appreciable degradation activity was observed in the algal, plankton, surface film, and microorganism cultures.

72-1963. Deichmann, W. B.; MacDonald, W. E.; Cubit, D. A.; Beasley, A. G. (Res. and Teaching Center of Toxicol., Dept. of Pharmacol., U. of Miami, Sch. of Med., P.O. Box 8216, Coral Gables, FL 33124). **Effects of starvation in rats with elevated DDT and dieldrin tissue levels.** *Int. Arch. Arbeitsmed. 29: 233-252; 1972.* (9 references)

To study changes in pesticide storage during starvation, groups of 12 male and 12 female weanling, adult, or old Osborne-Mendel rats were fed standard laboratory diets supplemented with 50 ppm *p,p'*-DDT, 7.5 ppm aldrin, or both of these. Half of each group was sacrificed after 4 wk; the other half was starved for 6 days, then sacrificed. Total DDT and dieldrin concentrations in the blood were reduced in all groups as a result of starvation. Both the concentration and the total quantity of total DDT and dieldrin in the liver were reduced after starvation, except in adult and old females. Starvation resulted in marked conversion of DDT to DDD (TDE) in the liver, especially in males. The total body burden of DDT and its derivatives as well as dieldrin decreased during severe starvation, especially in weanlings. Moderate-to-marked increases in the concentrations of DDT, its metabolites, and dieldrin in the abdominal fat of females occurred during starvation, whereas in the males, total DDT increased but dieldrin decreased. Additional findings were enhanced hepatic microsomal enzyme activity in starved rats, and increased weight gains in the groups of rats on the pesticide-supplemented diets.

72-1964. Feaster, J. P.; Van Middelem, C. H.; Davis, G. K. (Dept. of Animal Sci., U. of Florida, Gainesville, FL 32601). **Zinc-DDT interrelationships in growth and reproduction in the rat.** *J. Nutr. 102(4): 523-527; 1972.* (18 references)

The effects of maximum permissible human dietary levels (FDA regulations) of DDT, parathion, and carbaryl on growth and reproduction were investigated in rats subjected to the stresses of pregnancy and low and high levels of zinc. Weight gain was normal with the low level of zinc (10 ppm) or with pesticides, but high levels of zinc, with or without pesticides, produced a significant decrease in weight gain. Zinc uptake by red cells, and lactic dehydrogenase activity were unaffected by pesticides but affected by zinc levels. A high zinc level with pesticide resulted in a reduction in maternal and fetal hemoglobin. Low zinc diets did not affect reproduction. High zinc diets with or without pesticides resulted in smaller fetuses and a higher incidence of uterine deaths. High zinc diets produced liver concentrations of zinc 10 times those obtained with adequate or low zinc diets. Meaningful tissue pesticide levels were only obtained with DDT. Added dietary DDT produced total DDT levels 4 to 80 times higher in fat and 2 to 25 times higher in liver than amounts of DDT normally found in diet produced. High zinc increased the uptake of DDT from diets in which there was no additional DDT. Fetal livers were found to contain DDT.

72-1965. Oki, K. (Hayashigakuen Junior Women's Coll., Japan). **[A study on the molecular electronic structure of environmental contaminants.]** *Kagaku No Ryoiki (J. Jap. Chem.) 25(12): 1146-1152; 1971.* (6 references) (Japanese)

The electronic structures of some important organochlorine pesticides and other environmental pollutants are discussed in relation to their metabolism and toxicity. The history of DDT and the discovery of its toxicity, as well as findings on the toxicity of β-BHC, lindane, and dieldrin are reviewed briefly. Possible electronic orbital configurations of the chlorine atom are illustrated. The bond which is responsible for the degradation of organochlorine compounds and the production of free radicals is suggested as the agent responsible for their acute toxicity. The C-C double bond may also be a source of harmful effects, although it rarely occurs in pesticides. In structures containing $d\pi$-$p\pi$ orbitals, the toxicity of the *d* electron is problematic. The use of compounds containing π-orbital electrons is suggested as a substitute. Examples of such compounds include the recently developed carbamates, which in the future may be used in various combinations with amino acids, aliphatic acids, sugars, and nucleic acids in order to reduce harmful effects on desirable species.

72-1966. Plakhova, L. G. (Tashkent Med. Inst., Dept. of Hyg., Tashkent, USSR). **Sravnitel'naya toksichnost' gerbitsidov i profilaktika otravleniy. [Poisoning, prophylaxis and comparative toxicity of herbicides.]** *Med. Zh. Uzb. 8: 30-33; 1971.* (Russian)

The toxicity of cotoran (fluometuron), treflan (trifluralin), linuron, and erbon was studied in guinea pigs, white mice, rats, and rabbits to develop criteria for occupational prophylaxis. Fluometuron produced general poisoning; its oral LD50 was 810 mg/kg, the single threshold for inhalation was 558 mg/m^3. Chronic inhalation exposure for 4 mo produced a 30% increase in the leukocyte count as well as altering serum cholinesterase, catalase, and transaminase; impairment of the liver carbohydrate function and effects on the thyroid were also observed. The threshold for chronic inhalation exposure to fluometuron was established at 28 mg/m^3 Inhalation exposure to trifluralin in doses from 5000 to 7000 mg/m^3 produced no deaths among the experimental animals but purulent conjunctivitis was observed to last five or seven days before complete recovery. Acute poisoning was induced only by intragastric administration of fluometuron; its LD50 ranged from 2300 mg/kg in mice to 4000 mg/kg in rats. The oral and inhalational LD50 of linuron in the most susceptible animals was 1025 and 346 mg/kg respectively. The LD50 of erbon was 1023 mg/kg po, and 3014 mg/kg when applied topically to intact skin. Despite the low toxicity of these herbicides, it is recommended that care be taken while handling them.

72-1967. Araki, S. (School of Legal Med., Fac. of Med., Kumamoto U., Kumamoto-shi, Japan). **[Studies on organofluorine pesticides from the medico-legal viewpoint.]** *Nippon Hoigaku Zasshi (Jap. J. Leg. Med.) 26(3): 203-219; 1972.* (37 references) (Japanese)

Forensic studies were performed on Furatol (rodenticide, 1% aqueous solution of sodium monofluoroacetate); Fussol (acaricide, 10% aqueous solution of monofluoroacetamide); and Nissol (acaricide, 25% emulsifiable concentrate of *N*-methyl-*N*-(1-naphthyl)-monofluoroacetamide, or MNFA). The acute LD50 in dd-k strain mice was 13.5 mg/kg (oral) and 16.13 mg/kg(sc) for Furatol, 30.62 (oral) and 34.20 mg/kg (sc) for Fussol, and 27.42 (oral) and 28.58 mg/kg (sc) for Nissol. The principal toxic symptoms included listlessness, irritability, clonic convulsions, abasia, piloerection, and irregular respiration. Furatol had the greatest subacute toxicity, while the toxicities of Fussol and Nissol were similar. Subacute toxic symptoms included emaciation, anorexia, alopecia, abscess formation, and cyanosis. Qualitative assay methods suitable for these compounds included spot tests, detection of fluorine as hydrogen fluoride, paper chromatography, and thin-layer chromatography; however, UV assay was unsuitable. Sodium monofluoroacetate was detectable in the organs of rabbits killed by administration of 10 mg/kg of Furatol. Quantitative analysis of monofluoroacetate by the colorimetric method revealed that 19.7% was present in the stomach contents, 3.2% in liver, 1.2% in kidney, and 1.1% in blood. Monofluoroacetate was not detectable in brain or urine.

72-1968. Takagi, S. (Okayama U. Med. Sch., Dept. of Internal Med., Okayama, Japan). **[Studies on agricultural organofluoride poisoning. Part I. Effects of organofluorides on carbohydrate metabolism, liver function, and serum electrolytes.]** *Okayama Igakkai Zasshi (J. Okayama Med. Soc.) 81(11-12): 623-631; 1971.* (Japanese)

The effects of acute organofluoride poisoning on carbohydrate metabolism, liver function, and serum electrolytes were investigated in rabbits and rats. After the rabbits had been fasted for 24 hr, they were given sc injections of a 25% solution of Nissol (MNFA); blood samples were taken 30 min. before and every 30 min. after the injection. Fussol (10% solution) was administered orally to rats at 50 mg/kg body weight. Fluctuations in liver G-6-phosphate dehydrogenase were observed 3 or 4 hr after the administration. Rabbits showed no consistent abnormalities in the blood glucose levels, but hypoglycemia was recorded in rats, especially before and after the onset of convulsions. The lactate dehydrogenase system displayed secondary augmentation due to blockage of the Krebs cycle. No abnormalities were observed in the serum transaminases and colloidal reactions. No significant changes were noted in serum Na, K, Ca, and Cl.

72-1969. Takagi, S. (Okayama U. Med. Sch., Dept. Internal Med., Okayama, Japan). **[Studies on agricultural organofluoride poisoning, Part III. Influence of an organofluoride on the electrocardiogram.]** *Okayama Igakkai Zasshi (J. Okayama Med. Soc.) 81(11/12): 641-650; 1971.* (42 references) (Japanese)

The effects of Nissol (MNFA) on the heart were studied, based on electrocardiograms recorded in healthy experimental rabbits and in five chronic poisoning patients. All of the patients were male, between 21 and 35 years of age, and were poisoned while spraying diluted Nissol. The main symptoms were nausea, weakness, and fatigue, but one case showed profound disturbance of consciousness, and was almost unable to talk. For the rabbit tests, the 25% Nissol emulsion was diluted 1:10 with distilled water, and 1 ml of the diluted solution administered orally or by subcutaneous injection. The rabbit electrocardiograms showed initial depression of the ST segments, with gradual development of giant T waves, and occurrence of ventricular flutter and fibrillation immediately before death. The ECG tracings from the chronic poisoning patients showed bradycardia, PQ prolongation, and U waves, but no detectable arrhythmia. These phenomena were consistent with the assumption of vagotonia or hypokalemia.

72-1970. Canlorbe, P.; Bader, J.-C.; Job, J.-C. (Hopital St. Vincent de Paul, Paris, France). **Problemes diagnostiques poses par une tumeur virilisante maligne de la cortico-surrenale. (A propos d'une observation – Revue de la litterature). [Diagnostic problems arising from a virilizing tumor of the adrenal cortex. (Case report – Literature review).]** *Sem. Hop. 47(39): 2255-2270; 1971.* (28 references) (French)

The use of *o,p'*-DDD (TDE) in the treatment of malignant tumors of the adrenal cortex is discussed and reviewed. Reference is made to 138 cases treated with *o,p'*-DDD; a 50% drop in urinary 17-ketosteroids was found in 60% of the patients, while considerable regression of the clinical symptoms was observed in 34% of the patients. However, these improvements lasted an average of only seven months, postponing death by approximately one year. Treatment with *o,p'*-DDD may induce adrenal insufficiency. Its use is suggested only in cases where surgery failed or in which surgery is no longer feasible due to metastases or other signs of advanced disease conditions. Along with amino glutethimide, *o,p'*-DDD may be the only means of prolonging life since the tumors of the adrenal cortex are resistant to radiation treatment.

72-1971. Nishimura, H.; Tanimura, T. (Dept. of Anatomy, Faculty of Med., Kyoto Univ., Kyoto-shi, Japan). **[Drugs and congenital anomalies of the central nervous system.]** *Shinkei Kenkyu No Shimpo (Adv. Neurol. Sci.) 16(2): 310-322; 1972.* (76 references) (Japanese)

Recent findings regarding the production of congenital anomalies in the central nervous system by drugs and other chemicals are reviewed. Congenital CNS and other anomalies in humans reportedly attributable to ingestion of one of 32 drugs by the mothers are tabulated. The congenital anomalies observed in mammalian offspring after administration of various chemicals to the mothers during pregnancy are also listed. Anophthalmia and microphthalmia have been recorded after administration of chlormequat chloride and sodium arsenate in the hamster and after administration of apholate in the sheep. Chlormequat chloride and sodium arsenate also produced encephalocele in the hamster, and sodium arsenate produced exencephaly. Exencephaly in the mouse was produced by phenylmercuric acetate and thio-TEPA. Malathion was among the compounds which produced hydrocephaly in the rat. Continued studies on mutagenicity and teratogenicity of chemicals are needed, especially in view of the extended susceptibility period of the developing offspring to CNS anomalies. Abnormalities in retina, cerebral cortex, and cerebellum may even be produced by contact with chemicals during the postnatal period.

72-1972. Hollaender, A. (Ed.) (Div. of Biology, Oak Ridge National Lab., Oak Ridge, TN). **Chemical mutagens. Principles and methods for their detection. Vol. 1 and 2.** *Plenum Press, New York-London, 1971. 610 pp.* (3082 references)

Sponsored by the Environmental Mutagen Society, this book is designed to be used by investigators concerned with the field of mutagenesis as a guide to available study techniques and results. Volume 1 has a theoretical orientation, beginning with a discussion of the molecular mechanisms of mutations and continuing with presentations of methods for detecting mutagenic effects on DNA. Mutagen screening techniques employing bacteriophage, bacteria, and yeasts are also described here, and the relationships between mutagenicity, carcinogenicity and teratogenicity outlined. The second volume deals with some specific techniques for detection of mutagenesis in higher plants and animals, as well as in plant root tips and mammalian cell cultures. The needs and available techniques for a human population monitoring system are presented. The editor concludes that for the present at least, a battery of tests will be needed to give a reasonably comprehensive picture of the mutagenic capabilities of a compound, and that the three procedures previously recommended for mutagenicity testing meet the criteria for practical applicability. These are the host-mediated assay, the dominant-lethal test, and the *in vitro* chromosome aberration test.

72-1973. Humiczewska, M.; Stanosz, S. (Dept. of Biol., Med. Acad., Szczecin, Poland). **The effect of the phenoxyacetic acid derivatives on the enzymatic activity of the placenta in guinea-pigs.** *Folia Histochem. Cytochem. 9(1): 77-94; 1971.* (24 references)

Histochemical studies were performed on 25 placentas from guinea pigs, treated from the second day of gestation with subcutaneous injection of Pielik (2,4-D sodium salt) at 15 or 30 mg/kg/day; Chwastoks (dimethyl-4-chlorophenoxyacetic acid sodium salt) at 30 mg/kg/day; or Nitrogranulogen ([*N*-methyl] bis-β-chloroethylamine) at 20, 40, or 60 mg/kg/day. The alkaline phosphatase activity in the low-dose Pielik group and the Chwastoks group was lower than that in the control, and remained at a uniform level throughout gestation; in the high-dosage Pielik group, it was higher than in the control, but as in the control, it fluctuated throughout the gestation period. Higher acid phosphatase levels were found in the labyrinth zone of the placenta as compared with the spongy zone in control animals, whereas in the experimental groups, the spongy zone showed a higher acid phosphatase content, and no acid phosphatase was detected at all in the labyrinth zone of the lower-dosage Pielik and the Nitrogranulogen-treated groups. The higher-dosage Pielik group and the Chwastoks group demonstrated a rise in succinate dehydrogenase activity, and the others a fall as compared with the control group.

72-1974. Bajgar, J. (Purkyne Med. Res. Inst., Hradec Kralove, Czechoslovakia). **Inhibition of acetylcholinesterase in different parts of the rat brain by isopropyl methylphosphonofluoridate; *in vitro* and *in vivo* experiments.** *Biochem. Pharmacol. 21(5): 687-694; 1972.* (15 references)

Inhibition of AChE in the cerebellum, pons, frontal cortex and basal ganglia of the rat brain by isopropyl methylphosphonofluoridate (sarin) *in vitro* was studied and constants characterizing this inhibition (I_{50} and bimolecular rate constant, k*a*) were determined. They were practically identical for all parts of the brain studied. In experiments *in vivo*, the dependence of AChE inhibition on the dose of organophosphate, and the time course of AChE inhibition following dose of 0.25 mg/kg im were studied. The dose required for 50 per cent inhibition of AChE (I_{50} dose) was the highest for the basal ganglia and the lowest for the frontal cortex and the cerebellum. The rate of AChE inhibition *in vivo* was most rapid in the frontal cortex and slowest in the basal ganglia. A correlation between AChE activity in different parts of the brain and I_{50} dose of these parts was demonstrated. The differences in inhibition produced in the various areas of the brain are probably caused by different AChE concentration in these structures. (Author abstract by permission)

72-1975. Patocka, J.; Bielavsky, J. (Purkyne Med. Res. Inst., Hradec Kralove, Czechoslovakia). **Reactivation of isopropyl-methylphosphonylated acetylcholinesterase by α,ω-bis-(4-hydroxyiminomethylpyridinium)-2-*trans*-butene dibromide -- the effect of pH.** *Biochem. Pharma-*

col. 21(5): 742-745; 1972. (8 references)

The influence of pH on the reactivation by α,ω-bis-(4-hydroxyiminomethylpyridinium)-2-*trans*-butene dibromide of bovine erythrocyte AChE inhibited by sarin was investigated. The reactivator-inhibited enzyme complex was found to be more stable at higher pH, and the kinetic constants for both the formation of the intermediate and the dissociation resulting in reactivated enzyme are pH dependent. The pH optimum was calculated to be 7.5 from constants obtained from the pH profile, while the experimental value obtained was 7.25. Other authors have observed slight differences between observed and calculated values of pH optimum for oxime reactivation of organophosphate-inhibited AChE.

72-1976. Gawhary, A. S. (57 Esmond St., Liverpool L6 5AY, Lancs., England). **The effects of 2,2-bis (*para* chloro-phenyl)1,1-dichloroethane (DDD) on choline acetylase of the thymus gland.** *Biochem. Pharmacol. 21(5): 887-890; 1972.* (5 references)

In the rabbit, the commercial grade 2,2-bis(*p*-chlorophenyl)1,1-dichloroethane (DDD, TDE), caused acute atrophy of the thymus gland and hypertrophy of the adrenals. The choline acetylase activity of these thymus glands showed significant difference from those treated with *m,p'* isomer of DDD which caused hypertrophy of the thymus gland with increased choline acetylase (ChA) activity. (Author abstract by permission)

72-1977. Welsch, F.; Dettbarn, W.-D.* (Dept. of Pharmacol., Vanderbilt U. Sch. of Med., Nashville, TN 37203). **Inhibition of cholinesterases of rat diaphragm muscle by organophosphates and spontaneous recovery of enzyme activity *in vitro*.** *Biochem. Pharmacol. 21(5): 1039-1049; 1972.* (35 references)

Pretreatment of the isolated rat diaphragm with organophosphorus compounds, diisopropyl phosphorofluoridate (DFP), paraoxon or phospholine, resulted in inhibition of cholinesterase activity. Spontaneous recovery occurred after washing in Locke's solution for various periods of time (15-180 min). The rate of reactivation was fastest after inhibition with paraoxon and slowest after DFP and was significantly faster for butyrylcholinesterase than for acetylcholinesterase. Similar experiments when performed with the peripheral nerve of lobster, a tissue high in cholinesterase, showed no spontaneous recovery of enzyme activity, even after much longer periods of washing. (Author abstract by permission)

72-1978. Desaiah, D.;. Cutkomp, L. K.; Yap, H. H.; Koch, R. B. (Dept. of Entomol., Fisheries and Wildlife, U. of Minnesota, St. Paul, MN 55101). **Inhibition of oligomycin-sensitive and -insensitive magnesium adenosine triphosphatase activity in fish by polychlorinated biphenyls.** *Biochem. Pharmacol. 21(6): 857-865; 1972.* (37 references)

Tests *in vitro* with four polychlorinated biphenyls (PCBs) on four tissues of fish showed prominent inhibitory effects on oligomycin-insensitive Mg^{2+} ATPase, with muscle homogenate being most sensitive. Aroclors 1242 and 1254, in the intermediate range of chlorination, were more effective than 1221 and 1268. Mg^{2+} ATPase from mitochondria was not as sensitive to the PCBs when compared with DDT-type compounds which were more effective on mitochondrial Mg^{2+} ATPase than on oligomycin-insensitive Mg^{2+} ATPase. Some stimulation of Mg^{2+} ATPase was evident from the poorest inhibitors, Aroclors 1221 and 1268. Na^{+}-K^{+} ATPase from fish brain homogenate was inhibited by Aroclor 1242 but the dose required was several times that for Mg^{2+} ATPase. (Author abstract by permission)

72-1979. Mull, R. L.; Giri, S. N. (Dept. of Physiol. Sci., Sch. of Vet. Med., U. of California, Davis, CA). **The role of renal aromatic *N*-deacetylase in selective toxicity of avicide 3-chloro-*p*-toluidine in birds.** *Biochim. Biophys. Acta 273(1): 222-228; 1972.* (10 references)

Several species of birds were studied to determine the amount of *N*-deacetylase activity present in the kidneys. This information was desired to learn if there was any apparent relation between the presence of the enzyme and the selective nephrotoxicity of the avicide 3-chloro-*p*-toluidine. The active renal enzyme was absent from the hawk kidney, but was present in the kidneys of all other birds studied. The resistance of the hawk to 3-chloro-*p*-toluidine and the absence of the enzyme from its kidneys indicate that the deacetylase is probably involved in the nephrotoxicity of the avicide 3-chloro-*p*-toluidine. No comparable activity was found in the rat kidney or liver, or in the chicken liver. (Author abstract by permission)

72-1980. Hirwe, A. S.; Metcalf, R. L.*; Kapoor, I. P. (Depts. of Entomol. and Zool., U. of Illinois, Urbana, IL 61801). **α-Trichloromethylbenzylanilines and α-trichloromethylbenzyl phenyl ethers with DDT-like insecticidal action.** *J. Agr. Food Chem. 20(4): 818-824; 1972.* (12 references)

A series of *p,p'*-disubstituted α-trichloromethylbenzylanilines which closely resembly DDT-type compounds in structure have been synthesized and evaluated for insecticidal activity. These compounds affect insects in a manner indistinguishable from DDT, and the most effective compounds such as α-trichloromethyl-*p*-ethoxybenzyl-*p*-methylaniline were of comparable toxicity with DDT to flies and mosquito larvae. The metabolism of ^{3}H-α-trichloromethyl-*p*-ethoxybenzyl-*p*-ethoxyaniline was studied in the housefly, salt-marsh caterpillar, mouse, and in a model ecosystem where the compound was substantially biodegradable. A compar-

able series of α-trichloromethylbenzyl phenyl ethers was also shown to have DDT-like action. (Author abstract reprinted by permission of the American Chemical Society)

72-1981. Richey, Jr., F. A.; Bartley, W. J.; Fitzpatrick, J. T.; Kurtz, A. P. (Res. and Develop. Div., Union Carbide Corp., South Charleston, WV 25303). **Chemical synthesis of the carbaryl metabolite *trans*-5,6-dihydro-5,6-dihydroxy-1-naphthyl methylcarbamate.** *J. Agr. Food Chem. 20(4): 825-828; 1972.* (18 references)

Multigram quantities of *trans*-5,6-dihydro-5,6-dihydroxy-1-naphthyl methylcarbamate have been prepared from 1,5-dihydroxynaphthalene in three steps. The synthetic material was shown to have the indicated structure by interpretation of its mass, infrared, nmr, and ultraviolet spectra and was shown to co-crystallize with a radioactive sample of carbaryl metabolite "B" isolated from cow's urine. *trans*-5,6-Dihydro-5,6-dihydroxy-1-naphthol (carbaryl metabolite "D") has also been prepared. (Author abstract reprinted by permission of the American Chemical Society)

72-1982. Pfaender, F. K.; Alexander, M.* (Lab. of Soil Microbiol., Cornell U., Ithaca, NY 14850). **Extensive microbial degradation of DDT *in vitro* and DDT metabolism by natural communities.** *J. Agr. Food Chem. 20(4): 842-846; 1972.* (19 references)

Extracts of *Hydrogenomonas* sp. cells converted 1,1,1-trichloro-2,2-bis(*p*-chlorophenyl)ethane (DDT) to 1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethane (DDD, TDE), 1-chloro-2,2-bis(*p*-chlorophenyl)ethane (DDMS), 4,4′-dichlorobenzophenone (DBP), and several other products under anaerobic conditions. *p*-Chlorophenylacetic acid was formed when whole cells and oxygen were subsequently added, thereby demonstrating that enzymes of a single organism can convert DDT to ring-cleavage products. A strain of *Arthrobacter* grew on *p*-chlorophenylacetic acid, thus showing that action of two bacteria leads to extensive biodegradation of DDT. The *Arthrobacter* formed *p*-chlorophenylglycolaldehyde and other products from *p*-chlorophenylacetic acid. The major metabolites formed by microbial communities of sewage and fresh water containing sediment were DDD and DBP, but small amounts of DDMS and 1,1-dichloro-2,2-bis(*p*-chlorophenyl)ethylene and no *p*-chlorophenylacetic acid accumulated. Added *p*-chlorophenylacetic acid was rapidly decomposed by the sewage microflora. (Author abstract reprinted by permission of the American Chemical Society)

72-1983. Locke, R. K.; Baron, R. L. (Div. of Pestic. Chem. and Toxicol., Food and Drug Adm., Dept. of Health, Education, and Welfare, Washington, DC 20204). **Preforan metabolism by tobacco cells in suspension culture.** *J. Agr. Food Chem. 20(4): 861-867; 1972.* (21 references)

Preforan (*p*-nitrophenyl-α,α,α-trifluoro-2-nitro-*p*-tolyl ether, fluorodifen), labeled with ^{14}C in the C_1 position of the 4-nitrophenyl moiety or in the CF_3 group, was introduced into the medium of tobacco cells in suspension culture. After 15 days of incubation, recovery of added radioactivity varied between 52 and 76%. Of the recovered radioactivity, 60 to 80% was incorporated into the cells, with the remainder appearing in the medium or cell wash. No unchanged parent compound could be detected in any fraction. Radiolabeled Preforan incubated in cell-free medium was recovered unchanged. Metabolites present in cells and culture medium produced from $^{14}C_1$-labeled Preforan were characterized as conjugates of 4-nitrophenol, including probable glucoside and amino acid or protein conjugates, together with unidentified acidic conjugates. $^{14}C_1$-labeled 4-nitrophenol appeared in the medium but not in the cells. Metabolites present in cells and medium produced from $^{14}CF_3$-labeled Preforan probably represent ^{14}C incorporation into natural products, resulting from oxidation and cleavage of the $^{14}CF_3$ group from the parent compound. (Author abstract reprinted by permission of the American Chemical Society)

72-1984. Kaufman, D. D.; Plimmer, J. R.; Iwan, J.; Klingebiel, U. I. (Plant. Sci. Res. Div., ARS, U.S. Dept. of Agr., Beltsville, MD 20705). **3,3′,4,4′-Tetrachloroazoxybenzene from 3,4-dichloroaniline in microbial culture.** *J. Agr. Food Chem. 20(4): 916-919; 1972.* (24 references)

3,3′,4,4′-Tetrachloroazoxybenzene was isolated from cultures of *Fusarium oxysporum* Schlecht growing in the presence of ^{14}C-ring-labeled 3,4-dichloroaniline (3,4-DCA). Its identity was established by comparison of its chemical and physical properties with a synthetic sample. Mass spectra, infrared, and ultraviolet spectral properties were determined. It is probable that azoxybenzene is formed by oxidative condensation of two molecules of 3,4-dichlorophenylhydroxylamine or by the condensation of 3,4-dichlorophenylhydroxylamine with 3,4-dichloronitrosobenzene which are potential intermediates in the oxidation of 3,4-dichloroaniline to 3,4-dichloronitrobenzene. (Author abstract reprinted by permission of the American Chemical Society)

72-1985. Hatfield, C. T.; Anderson, J. M. (Fisheries Res. Bd. of Canada Biol. Station, St. Andrews, N.B., Canada). **Effects of two insecticides on the vulnerability of Atlantic salmon (*Salmo salar*) parr to brook trout (*Salvelinus fontinalis*) predation.** *J. Fish. Res. Bd. Can. 29(1): 27-29; 1972.* (11 references)

A 24-hr exposure to 1.0 ppm of Sumithion (fenitrothion) resulted in a decrease in learning ability of the Atlantic salmon (*Salmo salar*) parr as demonstrated by its increased vulnerability to predation by brook trout (*Salvelinus fontinalis*). In each experiment more fish

were eaten in the pond containing both treated and control fish than in that containing only controls, and in the former pond 95% of the treated fish were eaten as compared to 58% of the untreated ones. No significant effect was observed when the Atlantic salmon were treated with Sumithion at 0.1 ppm or with DDT at 0.07 ppm.

72-1986. Carlson, A. R. (Environ. Protection Agency, Natl. Water Quality Lab., Duluth, MN 55804). **Effects of long-term exposure to carbaryl (Sevin) on survival, growth, and reproduction of the fathead minnow (*Pimephales promelas*).** *J. Fish. Res. Bd. Can. 29(5): 583-587; 1972.* (18 references)

Fathead minnows (*Pimephales promelas*) were exposed to carbaryl concentrations between 0.008 and 0.68 mg/l. for 9 mo or for one life cycle. At the highest carbaryl concentration, reproduction was prevented and survival impaired. The testes of male fish maintained in the presence of the highest carbaryl concentration contained motile sperm; the ovaries of females were flaccid and apparently undergoing resorption. Carbaryl exposure (0.68 mg/l.) appeared to increase mortality among larvae (from unexposed parents) within 30 days of hatching. The 96-hr median tolerance concentration (TL50) and the lethal threshold concentration (LTC) for 2-month-old fathead minnows were found to be 9.0 mg/l.; the maximum acceptable toxicant concentration (MATC) is between 0.21 and 0.68 mg/l., at a water hardness of 45.2 mg/l. and a pH of 7.5.

72-1987. Oshiba, K.; Kawakita, H. (Osaka Municipal Inst. for Hyg., Kita-ku, Osaka-shi, Japan). **[Interaction between toxicants and nutrition III. Distribution and deposition of β-BHC in rat tissues.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Jap.) 13(3): 184-188; 1972.* (12 references) (Japanese)

Five groups of female Sprague-Dawley rats were fed a standard diet spiked with 10 ppm β-BHC, and the organ and tissue concentrations of β-BHC determined after 1, 2, 4, 6, and 8 wk. No differences in body or organ weights were found between treated animals and controls. The β-BHC content of the liver increased continuously during the first 4 wk, to 50 μg/g tissue lipid, then remained practically constant during the last 4 wk of feeding. This contrasted with the behavior previously observed for the γ-BHC levels, which passed through a maximum at 30 μg/g at the end of the second week, then gradually dropped to 10 μg/g by the end of 8 wk of feeding. Trends for β-BHC in kidney, brain, and blood were similar to those in liver, but the adipose tissue content rose continuously throughout the feeding period. After 8 wk, the adipose tissue had the highest β-BHC content (38.7 μg/g fresh weight), followed in decreasing order by liver, lung, kidney, spleen, heart, and brain (0.45 μg/g). The β-BHC levels were higher than the corresponding γ-BHC levels except in brain and blood (blood β-BHC, 0.15 g/ml; γ-BHC, 0.20 g/ml). No macroscopic abnormalities were observed in the organs.

72-1988. Oshiba, K.; Kawakita, H. (Osaka Municipal Inst. for Hyg., Kita-ku, Osaka-shi, Japan). **[Interaction between toxicants and nutrition IV. Effect of lipid metabolism on reduction of BHC deposition in rat tissues.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Jap.) 13(3): 189-194; 1972.* (13 references) (Japanese)

Two groups of female rats were fed standard laboratory diets spiked with 10 ppm β-BHC for 28 days, or 100 ppm γ-BHC (lindane) for 14 days, to produce equilibrium tissue levels of BHC. Some of the rats were then changed to diets containing 6 or 30% fat and no BHC for 3 or 6 days, while others were fasted for 3 or 6 days. A third group was transferred to the BHC-free standard diet, and the animals were given one of three types of hormones which accelerate the mobilization of body fats: adrenalin, ACTH, and TSH. Determination of BHC isomers in organs and tissues of these animals revealed that 6-days' feeding of a fatty diet produced appreciable reductions in the γ-isomer content (53, 76, and 74% respectively) of liver, kidney, and brain, but was not effective in reducing the β-isomer content in these organs or the γ-isomer content in the blood. Starvation also reduced the BHC deposition in organs compared to the controls (continued on the pesticide-spiked diets), but the rats were severely exhausted. The storage of β-BHC in starved rats was reduced only in adipose tissue (by 35.8%), while appreciable increases in β-BHC levels were noted in blood, liver, kidney, and brain. Reductions in adipose tissue β-BHC levels in the hormone-treated group were due to both fat mobilization and transfer to the pesticide-free diet.

72-1989. Oshiba, K.; Kawakita, H. (Osaka Municipal Inst. for Hyg., Osaka-shi, Japan). **[Relationship between toxic substances and nutrition. VI. Absorption and excretion of benzene hexachloride.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Jap.) 13(3): 244-245; 1972.* (Japanese)

Groups of adult female mice were adapted to BHC ingestion by feeding *ad lib.* a diet containing 10 ppm of γ-BHC (lindane) or β-BHC for 10 days, or 10 ppm of lindane for 49 days. After adaptation, 5 μCi of ^{14}C-labeled lindane or β-BHC was added to the diet for one day, and the radioactivity excreted in urine and feces each day determined. The excretion of labelled BHC isomers was greatest on the first and second days of the experiment, then dropped off sharply. The excretion in the urine was always greater than that in the feces. A total of 12.2-13.0% of the ingested β-BHC was excreted in the urine and 5.4-5.8% in the feces during the first 10 days after ingestion. In the case of the γ-isomer, 48.5-52.0% was excreted in the urine and 20.8-22.2% in the feces within 10 days. Although the rate of absorption could not be determined accurately, about 95% of

the β-isomer and about 85% of the γ-isomer seems to be absorbed from the digestive tract within 5 days. After 49 days of adaptation to lindane by continuous administration, the rate of excretion via the urine was apparently increased, but not the excretion via the feces. Thus, enhanced excretion was the result of accelerated metabolism, not reduced absorption. This agrees with the previous observations, according to which the residual γ-BHC in the tissues slowly decreases after a certain time period.

72-1990. Schafer, E. W. (Wildlife Res. Center, Bureau of Sport Fisheries and Wildlife, U.S. Dept. of the Interior, Denver, CO 80225). **The acute oral toxicity of 369 pesticidal, pharmaceutical and other chemicals to wild birds.** *Toxicol. Appl. Pharmacol. 21(3): 315-330; 1972.* (5 references)

The acute po toxicity of 369 chemicals was determined for 1 or more species of wild birds, always including either red-winged blackbirds or starlings. Of these, 180 chemicals were toxic to 1 or more species at 100 mg/kg or less. Statistical comparison of redwing, starling, and rat data indicated that redwings were more sensitive to chemicals than starlings, and both were more sensitive than rats. (Author abstract by permission)

72-1991. Whitehead, C. C.; Pettigrew, R. J. (Agr. Res. Council's Poultry Res. Centre, West Mains Rd., Edinburgh, EH9 3JS, Scotland). **The subacute toxicity of 2,4-dichlorophenoxyacetic acid and 2,4,5-trichlorophenoxyacetic acid to chicks.** *Toxicol. Appl. Pharmacol. 21(3): 348-354; 1972.* (6 references)

Chicks were able to tolerate large dietary doses of the herbicides 2,4-dichlorophenoxyacetic acid (2,4-D) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), the only adverse effects being a reduction in food consumption and growth rate. A level of 5000 mg 2,4,5-T/kg proved fatal to chicks; a similar level of 2,4-D, while not resulting in death, did cause histological changes. Chicks were able to tolerate this level of either herbicide for up to 1 wk, however, and resume a normal growth rate when returned to uncontaminated food. No specific effect of high dietary levels of these herbicides was noted on plasma calcium or magnesium concentrations. The birds were able to discriminate between contaminated and uncontaminated food; when given a choice they rejected contaminated food and grew at a normal rate. (Author abstract by permission)

72-1992. McElligott, T. F. (Dept. of Pathol., Hotel Dieu Hosp., and Queen's U., Kingston, Ont., Canada). **The dermal toxicity of paraquat: differences due to techniques of application.** *Toxicol. Appl. Pharmacol. 21(3): 361-368; 1972.* (12 references)

The acute and subchronic dermal toxicity of paraquat was investigated in the rabbit by an occlusive technique and by one in which the site of application was exposed to the air. Toxicity was greatly reduced by air drying. Routine decontamination with water removed over 99.9% of the paraquat; however, when free grooming was allowed after washing the application site, residual skin contamination caused severe tongue ulceration and inability or unwillingness to eat. Tongue lesions and adverse effects were minimized by continued restraint even after decontamination. The subacute (20 day) dermal LD50 of paraquat ranged from 4.5 mg cation per kg body weight by the occlusive technique, to over 24 mg cation per kg body weight when air drying occurred and grooming was rigidly prevented. Using the latter technique animals were not killed by a single application of 480 mg cation/kg and systemic signs of intoxication were not evident. In contrast, a single application of 240 mg cation/kg was fatal within 72 hr when applied beneath an occlusive dressing. (Author abstract by permission)

72-1993. Weil, C. S.; Woodside, M. D.; Carpenter, C. P.; Smyth, Jr., H. F. (Mellon Inst., Carnegie-Mellon U., Pittsburgh, PA 15213). **Current status of tests of carbaryl for reproductive and teratogenic effect.** *Toxicol. Appl. Pharmacol. 21(3): 390-404; 1972.* (43 references)

A three-generation rat reproduction study with technical carbaryl (1-naphthyl methylcarbamate) added to the diet to provide daily doses up to 10 mg/kg resulted in no statistically significant, dose-related effect upon fertility, gestation, viability of pups, or lactation. A rat teratogenic study with carbaryl added to the diet to provide daily doses up to 500 mg/kg resulted in no increase in teratogenic anomalies and no effect on fertility or gestation. Only at the highest dose was weight gain reduced; many pups in this group died before weaning from carbaryl-fed mothers. A rat reproduction study included groups receiving carbaryl in the diet at daily doses up to 200 mg/kg, while other groups concurrently received carbaryl by po intubation of doses up to 100 mg/kg/day. A dose of 100 mg/kg/day given by intubation before mating resulted in mortality, signs of cholinesterase inhibition, and reduced fertility. None of these effects were seen in groups dosed in the diet. Reduction in growth at 100 mg/kg/day given by intubation was equivalent to that of 200 mg/kg/day given in the diet. Guidelines for an acceptable experimental design of studies to evaluate human risks are discussed in relation to published articles and unpublished reports on the effect of carbaryl on animal reproduction or teratology. Only one study other than the three reported here conformed to all the guidelines; in this study it was reported that no reproductive effects occurred in rats given 2000 ppm in the diet. (Author abstract by permission)

72-1994. Friedman, M. A.; Greene, E. J.; Csillag, R.; Epstein, S. S. (Dept. of Pharmacol., Med. Coll. of Va.,

Health Sci. Div., Va. Commonwealth U., Richmond, VA 23219). **Paradoxical effects of piperonyl butoxide on the kinetics of mouse liver microsomal enzyme activity.** *Toxicol. Appl. Pharmacol. 21(3): 419-427; 1972.* (18 references)

The kinetics of the effects of piperonyl butoxide on mouse liver aminopyrine demethylase, aniline hydroxylase, and biphenyl hydroxylases were investigated. Piperonyl butoxide competitively inhibited aminopyrine demethylase *in vitro* and *in vivo*, and aniline hydroxylase *in vitro*; however, *in vivo* inhibition of aniline hydroxylase was uncompetitive. Contrastingly, *in vitro* piperonyl butoxide noncompetitively inhibited biphenyl-4-hydroxylase, while competitively stimulating biphenyl-2-hydroxylase. Sulfoxide, a structurally related methylenedioxyphenyl derivative, also noncompetitively inhibited biphenyl-4-hydroxylase and competitively stimulated biphenyl-2-hydroxylase. Furthermore, when microsomal enzyme preparations from control and piperonyl butoxide-treated mice were mixed, aminopyrine demethylase and aniline hydroxylase activities were the same as the control alone, indicating that the inhibitory factors were enzyme bound. (Author abstract by permission)

72-1995. Khera, K. S.; McKinley, W. P. (Res. Labs., Food and Drug Directorate, Dept. of Natl. Health and Welfare, Ottawa, Ont., Canada). **Pre- and postnatal studies on 2,4,5-trichlorophenoxyacetic acid, 2,4-dichlorophenoxyacetic acid and their derivatives in rats.** *Toxicol. Appl. Pharmacol. 22(1): 14-28; 1972.* (11 references)

The herbicides 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) (containing less than 0.5 mg/kg of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin) and 2,4-dichlorophenoxyacetic acid (2,4-D) induced fetopathy and an increased incidence of skeletal anomalies following single daily po doses of 100-150 mg/kg on days 6-15 of gestation. Increased frequency of skeletal defects was also observed in single trials with butyl, isooctyl, butoxyethanol, and dimethylamine derivatives of 2,4-D. The observed skeletal defects did not appear to be incompatible with postnatal survival. Following treatment of dams with 2,4,5-T, 2,4 -D, and their butyl esters and the isooctyl ester of 2,4-D, weight gain and viability of the offspring were within control limits. The findings suggest that postnatal parameters were unrelated to the teratologic potential of the chemicals. (Author abstract by permission)

72-1996. Bajgar, J. (Purkyne Med. Res. Inst., Hradec Kralove, Czechoslovakia). **Inhibition of acetylcholinesterase in rat brain subcellular fractions following *O*-isopropyl methylphosphonofluoridate intoxication.** *Toxicol. Appl. Pharmacol. 22(1): 93-96; 1972.* (16 references)

Acetylcholinesterase inhibition in blood and subcellular fractions of the medulla oblongata of the rat brain was studied following *O*-isopropyl methylphosphonofluoridate (IMPF) intoxication. Total acetylcholinesterase inhibition at time of death was found only in microsomal and crude mitochondrial fractions (probably in nerve ending particles). (Author abstract by permission)

72-1997. Sullivan, L. J.; Chin, B. H.; Carpenter, C. P. (Chem. Hyg. Fellowship, Mellon Inst., Carnegie-Mellon U., 4400 Fifth Ave., Pittsburgh, PA 15213). ***In vitro vs in vivo* chromatographic profiles of carbaryl anionic metabolites in man and lower animals.** *Toxicol. Appl. Pharmacol. 22(2): 161-174; 1972.* (11 references)

An *in vitro* technique for metabolic studies utilizing liver tissue has been developed based upon successful organ maintenance methods and chromatographic profiles. This technique qualitatively reflects *in vivo* metabolic processes of animals, including man, under prescribed conditions without resorting to the risk of dosing human beings. This was accomplished by the utilization of the published literature on *in vivo* metabolism of carbaryl in rat, dog, guinea pig, and man. Under the required conditions, which simulate a dose level comparable to a chronic no-ill-effect feeding level in the rat, the variations between these *in vitro* and the previous *in vivo* results are substantially less than between species. (Author abstract by permission)

72-1998. Triolo, A. J.; Mata, E.; Coon, J. M. (Dept. of Pharmacol., Jefferson Med. Coll., Philadelphia, PA 19107). **Effect of acute ethanol intoxication on the distribution of dieldrin in mice.** *Toxicol. Appl. Pharmacol. 22(2): 201-207; 1972.* (14 references)

Ethanol (6.3 g/kg) or a calorically equivalent quantity of glucose was administered po to groups of adult male Swiss-Webster mice previously given a single po dose of dieldrin (16 mg/kg). Dieldrin levels were significantly elevated in plasma and liver 16 hr after ethanol administration. There was also an increase in total liver lipids 8 and 16 hr after ethanol. Although ethanol decreased the urine volume, there was no significant reduction in the total dieldrin excreted in the urine during the first 16 hr after ethanol. There was no difference between ethanol and glucose treated animals in respect to dieldrin levels in epididymal fat at 8, 16, and 24 hr after ethanol or glucose. Since ethanol did not decrease the concentration of dieldrin in epididymal fat, it appears that mobilization of dieldrin from storage sites in adipose tissue may not be primarily responsible for the observed increase in dieldrin levels in plasma and liver after ethanol. It is suggested that a decrease in urinary excretion of dieldrin following ethanol accounts for the increase in dieldrin levels in the tissues. (Author abstract by permission)

72-1999. Wolcott, R. M.; Vaughn, W. K.; Neal, R. A.* (Center in Environ. Toxicol., Dept. of Biochem., Vanderbilt U. Sch. of Med., Nashville, TN 37232). **Comparison of the mixed function oxidase-catalyzed metabolism of a series of dialkyl *p*-nitrophenyl phosphorothionates.** *Toxicol. Appl. Pharmacol. 22(2): 213-220; 1972.* (17 references)

Various concentrations of dimethyl, diethyl, di *n*-propyl and di *n*-butyl *p*-nitrophenyl phosphorothionate were incubated with rabbit liver microsomes, and the rates of formation of the corresponding dimethyl, diethyl, di *n*-propyl and di *n*-butyl *p*-nitrophenyl phosphates and dimethyl, diethyl, di *n*-propyl, and di *n*-butylphosphorothioic acids were determined. From these data the apparent *Km* and *Vmax* for the formation of each metabolite was calculated. The maximum rate of metabolism (*Vmax*) was found to vary with the size of the alkyl group. The maximal rate of metabolism of the dialkyl *p*-nitrophenyl phosphorothionates to the dialkyl *p*-nitrophenyl phosphates was exhibited by the di *n*-propyl and di *n*-butyl compounds. The maximal rate of metabolism to the dialkylphosphorothioic acids was shown by the dimethyl compound. A linear free energy plot failed to show a significant correlation between the polar effect of the alkyl groups (as quantitated by the Taft σ^* constant) or the alkoxy groups (as quantitated by the Kabachnik $\sigma\phi$ constant), and the maximal rates of metabolism of these compounds to either the dialkyl *p*-nitrophenyl phosphates or the dialkylphosphorothioic acids. Although the inductive and steric properties of the alkyl groups are undoubtedly important, there must be additional factors responsible for the differences seen in the rates of metabolism of this series of compounds. (Author abstract by permission)

72-2000. Sharp, C. W.; Ottolenghi, A.; Posner, H. S. (Natl. Inst. of Environ. Health. Sci., NIH, P.O. Box 12233, Research Triangle Park, NC 27709). **Correlation of paraquat toxicity with tissue concentrations and weight loss of the rat.** *Toxicol. Appl. Pharmacol. 22(2): 241-251; 1972.* (39 references)

Paraquat was administered to Sprague-Dawley and Wistar rats po and iv. Death generally occurred in 2-5 days, but some rats survived for up to 12.5 days. Loss of body weight correlated with lethality, and concentrations in lung and kidney correlated with the degree of toxic response as defined by 2 levels of weight loss and death. The lung became the organ of highest concentration of paraquat through an initial stasis phase, and paraquat was detected in the lungs of all rats that died. The initial half-life of paraquat in plasma and in the tissues was 20-30 min. The subsequent half-life of paraquat in muscle was longest, 4-5 days, and secondary half-lives in the plasma and other tissues were about 2 days. Diquat did not accumulate in lung and muscle as did paraquat, and did not produce the lung pathology. The accumulation in lung (critical organ of pathology), the relatively long secondary half-life of paraquat in the plasma and tissues, the correlation of tissue concentrations with degree of toxic response and the findings with diquat contradict the concept of paraquat as a "hit-and-run" poison. (Author abstract by permission)

72-2001. Matlib, M. A.; Kirkwood, R. C.; Patterson, J. D. E. (Dept. of Biol., U. of Strathclyde, Glasgow, Scotland). **Binding of certain substituted phenoxy-acids by bovine serum albumin.** *Weed Res. 11(2/3): 190-192; 1971.* (12 references)

In the study of oxidative phosphorylation and respiratory control in isolated mitochondria, bovine serum albumin (BSA) is frequently utilized to bind the liberated fatty acids and phenolic compounds which act as endogenous uncouplers. Several herbicides have been investigated as uncouplers of oxidative phosphorylation; in this study, the binding of these acids by BSA was investigated using a gel filtration method. Binding was identified by the appearance of a trough in the elution profile; the area of the trough was plotted against moles of herbicide added at a constant BSA concentration. The calculations indicated that 5 mol of MCPA, 2,4-D, or 2,4,5-T are required per mole of BSA to eliminate the trough, whereas 7 mol of the corresponding phenoxybutyric acid are required per mole of BSA. The mechanism of interaction between BSA and phenoxy herbicides is unknown at present.

72-2002. Grover, R. (Res. Sta., Canada Dept. of Agr., Regina, Sask., Canada). **Effect of picloram on some soil microbial activities.** *Weed Res. 12(1): 112-114; 1972.* (7 references)

Picloram-K salt was mixed with Regina heavy clay at 0, 0.25, 0.5, and 1 ppm (oven-dry soil); then soil samples were placed in pots, brought to field moisture capacity (41%), covered, and incubated at 25 ± 1°C for 172 days. Nitrate levels were determined colorimetrically by the phenol-disulfonic acid technique. Oxygen uptake was determined manometrically (Warburg apparatus), and carbon dioxide evolution was measured by the flask method. The nitrate content of the soil samples increased six to seven times during the incubation period, regardless of the picloram addition. Carbon dioxide evolution was not affected at any picloram concentration tested (up to 300 ppm), but oxygen uptake was enhanced at picloram concentrations of 100 and 300 ppm in the Warburg flasks. This indicates that picloram may have some effect on gross microbial metabolism in Regina heavy clay soil, but only at concentrations which far exceed those likely to be encountered in agricultural practice.

72-2003. Sparschu, G. L.; Dunn, F. L.; Lisowe, R. W.; Rowe, V. K. (Chem. Biol. Res., Dow Chem. Co., Midland, MI 48640). **Study on the effects of high levels of 2,4,5-trichlorophenoxyacetic acid on foetal development**

in the rat. *Food Cosmet. Toxicol. 9(4): 527-530; 1971.* (1 reference)

Commercial-grade 2,4,5-trichlorophenoxyacetic acid (2,4,5-T), containing 0.5 ppm 2,3,7,8-tetrachlorodibenzo-*p*-dioxin, did not produce a teratogenic response when administered orally to rats on days 6-15 of gestation at a dosage level of 50 mg/kg/day. In the fetuses, minimal effects in the form of a slightly higher incidence of delayed ossification of the skull bones were observed. 2,4,5-T given on days 6-10 of gestation at a level of 100 mg/kg/day was toxic to the dams and caused a high incidence of maternal deaths. Four of 25 pregnant rats survived. Of these, three had complete early resorptions and one had a litter of 13 viable fetuses which showed toxic effects but no terata. (Author abstract by permission)

72-2004. Holder, G. M.; Ryan, A. J.; Watson, T. R.; Wiebe, L. I. (Pharmacy Dept., U. of Sydney, Sydney 2006, New South Wales, Australia). **A note on the excretion of 2,6-di-*tert*-butyl-4-nitrophenol in the rat.** *Food Cosmet. Toxicol. 9(4): 531-535; 1971.* (5 references)

The metabolism of the sterically-hindered phenol, 2,6-di-*tert*-butyl-4-nitrophenol, was examined in the rat after oral, intravenous, and intraperitoneal administration of ^{14}C-labeled and unlabeled material. Urinary, fecal and biliary routes of excretion were examined. Excretion in the feces and urine was also examined after treatment of the gut with neomycin. Results indicate that the gut microflora play a role in the metabolism of 2,6-di-*tert*-butyl-4-nitrophenol. (Author abstract by permission)

72-2005. Sobotka, T. (Div. of Pestic. Chem. and Toxicol., Food and Drug Admin., Dept. of Health, Educ., and Welfare, Washington, DC 20204). **Comparative effects of 60-day feeding of maneb and of ethylenethiourea on thyroid electrophoretic patterns of rats.** *Food Cosmet. Toxicol. 9(4): 537-540; 1971.* (7 references)

Groups of rats were fed maneb at dietary levels from 250 to 1500 ppm or ethylenethiourea (ETU) from 5 to 500 ppm for 60 days. After this time, both compounds had induced similar changes in the electrophoretic pattern of thyroid-tissue extracts. These changes were a decrease in the density of a band presumably representing thyroglobulin, an increase in the density of a band representing a non-iodinated protein, and a slight decrease in the density of a band representing an iodinated protein. After electrophoresis of the thyroid extracts from ETU-fed rats given ^{131}I 24 hr before sacrifice, three iodoprotein groups were found. The decrease in radioactivity in the thyroglobulin band with increasing dose of ETU paralleled the change in density of the band. (Author abstract by permission)

See also 72-1815 72-1840 72-1845 72-1846 72-1848 72-1853 72-1864 72-1865 72-1872 72-1873 72-1888 72-1896 72-2017

72-2006. Frei, R. W.; Belliveau, P. E. (Trace Analysis Res. Centre, Dept. of Chem., Dalhousie U., Halifax, N. S., Canada). **pH-Sensitive fluorogenic spray reagents. II. Quantitative studies with pesticides.** *Chromatographia 5: 296-300; 1972.* (4 references)

A quantitative method of detecting sulfur-containing pesticides on TLC plates was developed. The method utilized a spray of 1,2-dichloro-4,5-dicyanobenzoquinone, which produced a fluorescence that could be measured *in situ*. Changes in acidity affected the fluorescent species. Best results were obtained using home-made MN silica gel N plates. A 30-min exposure to UV light gave maximum fluorescence, and benzene was the most satisfactory of the spray solvents tested. The limits of detection were 0.03 μg of sulfur-containing organophosphate and 0.1 μg for a sulfur-containing triazine. The standard deviation was about 5%. Recovery of malathion from tap water by this method was 90%.

72-2007. Hempel, D.; Liebmann, R.; Hellwig, A. (Hygiene-Institut Magdeburg, Pflanzenschutzmittel-Forschung des VEB Fahlberg-List, Magdeburg, DDR). **Eine neue kolorimetrische Bestimmung von Toxaphen in Wasser. [A new colorimetric determination of toxaphene in water.]** *Fortschr. Wasserchem. Ihrer Grenzgeb. 13(3): 181-188; 1971.* (11 references) (German)

Toxaphene is advantageously used on blossoming crops, since it kills biting insects without harming bees; however, it is highly toxic to fish. A method for determining toxaphene residues in water was developed. The water was acidified with sulfuric acid and extracted with *n*-hexane. The organic phase was washed with concentrated sulfuric acid, then with chromosulfuric acid to remove interfering organic materials which may have been present in the polluted water, and then with distilled water. The hexane was evaporated, the residue taken up in acetone and transferred to a Schiff tube. The acetone was evaporated off and 1 ml of 70% aqueous pyridine added; the mixture was then heated for 30 min on a water bath at 100°C. After cooling, the tube contents were treated with 4 ml of an ethanol-water (5:1) mixture and 0.2 ml of 9 *N* NaOH. After the mixture had been shaken, 1 ml of ethyl cyanoacetate was added dropwise. The color which developed was read at 550 nm after 1 hr. The limit of detection in clean water was 0.015 ppm, and the recovery rate between 70 and 95%; in polluted water, 0.05 ppm could be detected. DDT did not undergo this color reaction, but lindane did, so that the lindane value must be independently determined and subtracted from the toxaphene value found.

72-2008. Safe, S.; Hutzinger, O. (Atlantic Regional Lab., Natl. Res. Council of Canada, Halifax, N. S. Canada). **The mass spectra of polychlorinated biphenyls.** *J. Chem. Soc. 5: 686-691; 1972.* (31 references)

Analysis of several di- and tetra-chlorobiphenyls by mass spectrometry indicated randomization of chlorine over both phenyl rings prior to fragmentation in all but those compounds having two or more chlorine atoms *ortho* to the Ph-Ph bond. The metastable ion intensities resulting from the $M \rightarrow M-Cl_2$ reaction in the exceptive compounds are quite different from those of the other isomers. Most of the isomers exhibited similar primary ion spectra with successive losses of $Cl\cdot$ from the molecular ion. An important consideration in the analysis of polychlorinated biphenyls (PCB) is that in most cases primary ion mass spectra do not distinguish between those PCB isomers which are inseparable by GLC. In the lower chlorine homologues, HCl may be released, but only reactions which expelled $Cl\cdot$ were accompanied by metastable ions. A similar pattern of fragmentation was suggested by information obtained from ion kinetic energy spectra.

72-2009. Fujita, M.; Sakagami, Y. (Dept. of Public Health Pharmaceutics, the Inst. of Public Health, Tokyo, Japan). **Study on the determination of residual amounts of ethylene bis-dithiocarbamates (agricultural fungicides).** *Koshu Eiseiin Kenkyu Hokoku (Bull. Inst. Pub. Health (Tokyo)) 19(3): 207-211; 1970.* (6 references)

A new method for determination of ethylene bis-dithiocarbamate is based on the measurement of ethylenediamine, one of its acid decomposition products. Sodium hydroxide was used to extract the fungicide from vegetables. The pH of the extract was brought to 5.0, and zinc chloride was added. The resulting precipitate was dissolved in internal standard solution, heated, and analyzed by GLC using a hydrogen flame detector. The alkali-modified chromosorb support phase was coated with Carbowax or dioctylphthalate. The presence of dialkyldithiocarbamates and thiram did not interfere with the analysis of ethylene bis-dithiocarbamate.

72-2010. Wohlgemuth, R. (Biologische Bundesanstalt fuer Land- und Forstwirtschaft, Institut fuer Vorratsschutz, Berlin-Dahlem, Germany). **Methoden zur Konzentrationsmessung von Methylbromid im Vorratsschutz. [Methods for measuring the concentration of methyl bromide in the protection of stored goods.]** *Mitt. Biol. Bundesanst. Land- Forstwirt. Berlin-Dahlem 142: 1-33; 1971.* (26 references) (German)

Methods and instruments suitable for determining the concentrations of gaseous methyl bromide in containers of grain, copra, etc., are discussed. Three physical methods of determining the methyl bromide concentration were represented by instruments suitable for field use in fumigation practice: thermal conductivity measurements, measurement of the heat of combustion over a heated wire, and optical refraction measurements. Chemical measurement methods were based on the change in a color indicator brought into

contact with methyl bromide. The water vapor and carbon dioxide contents of the air had a greater influence on the results obtained by thermal conductivity and optical refraction methods than by the interferometric and chemical methods; however, the accuracy of the first two methods was higher. The instrument used for interferometric methods was especially portable, could be used even in complete darkness, and in contrast to the thermal conductivity and heat of conduction instruments, was insensitive to movement, so that it could even be used in moving barges. In addition, it was the safest type of instrument in explosive atmospheres. A test of the suitability of various plastic tubing types for the collection of methyl bromide/air samples revealed that polyamide tubing had low methyl bromide absorption.

72-2011. Garty, O. M. (Analytical Group, Union Carbide Corp., Bound Brook, NJ). **Improved test for PCBs.** *Mod. Packag. 45(4): 46-50; 1972.* (12 references)

A combination of techniques was developed for determining polychlorinated biphenyls (PCBs) in polyethylene resins and products derived from them. Using all glass equipment, samples were extracted in nanograde hexane and passed through a Florisil adsorption column. A concentrate of the eluate was analyzed by gas chromatography using electron-capture and flame ionization detectors. The concentrate was also analyzed by neutron activation for Cl and Na. Infrared spectrometry was used to demonstrate the oxygenated nature of observed electron-capture peaks. The method indicated that if any PCBs were present in the base resin tested, the level would have to have been considerably less than 50 ppb and that the extruded food film containing the resin was also essentially free of PCBs.

72-2012. Weinmann, W.; Finger, E. (Laboratorium fuer Chemische Mittelpruefung der Biologischen Bundesanstalt fuer Land- und Forstwirtschaft, Braunschweig, Germany). **Analysenmethode zur potentiometrischen Bestimmung von Amitrol (3-Amino-1,2,4-triazol) in Pflanzenschutzmitteln. [Analytical method for potentiometric determination of amitrole (3-amino-1,2,4-triazole) in plant protection agents.]** *Nachrichtenbl. Deut. Pflanzenschutzdienstes 23(12): 184-188; 1971.* (6 references) (German)

A potentiometric method for determination of amitrole in various formulations is based on its precipitation with silver nitrate in buffered aqueous solution. Technical preparations and mixtures with other active ingredients, such as triazines, substituted ureas, growth hormones, bromacil, and trichloroacetic acid, were first extracted (Soxhlet) with anhydrous ethyl acetate. If ionizable chlorine alone was thought to be present, the ethyl acetate solution was taken directly for determination, using ammonium acetate as buffer and a silver/silver chloride or silver/mercurous sulfate electrode. In the case of mixtures with other chlorine-containing pesticides, the ethyl acetate was evaporated after extraction; the residue was dissolved in water and rinsed into the titration vessel with dilute acetic acid; then ammonium acetate was added and the titration performed with silver nitrate. The titration endpoint could be determined graphically or computationally, but the latter was preferred. The other ingredients mentioned did not interfere with the determination of amitrole under these conditions.

72-2013. Skrentny, R. F.; Dorough, H. W. (Dept. of Entomol., U. of Kentucky, Lexington, KY). **Efficiencies of several extraction procedures in removing organochlorine insecticides from tobacco.** *Tobacco Sci. 15: 111-113; 1971.* (13 references)

New and existing methods of extracting total DDT-TDE residues were compared using a single batch of cigarette tobacco. Extracts were chromatographed on a Florisil column and analyzed by GLC using a tritium electron-capture detector. The most efficient method involved a soxhlet extraction with a methanol:chloroform ratio of 9:1. Using this technique the average value for total DDT-TDE was 43 ppm or three times the quantity extracted by mechanical shaking in hexane and/or other solvents. Extractions carried out in a blender were of intermediate efficiency. Alkaline hydrolysis of the TDE residues was used to confirm identity of the material and also to allow detection of endrin. No endrin was found in the cigarette tobacco sampled.

72-2014. Maes, R. (Fakulteit Geneeskunde, Katholieke U. te Leuven, Louvain, Belgium). **Residubepaling van organisch gechloreerde koolwaterstofinsekticiden bij de mens. [Residue determination of organic chlorinated hydrocarbon insecticides in man.]** *Verh. Kon. Vlaam. Acad. Geneesk. Belg. 33(4): 352-395; 1971.* (78 references) (Flemish)

Many organochlorine insecticides are in use today, and while their acute toxicity is low, the possibility of chronic effects due to accumulation in human tissues, especially in adipose tissue, must be kept in mind. Methods of organochlorine residue determination in man are reviewed. TLC was often used, especially for screening, because of its speed, simplicity, and sensitivity. Activated silica gel G plates and an isooctane/acetone (98:2) solvent system gave good separation of the various pesticides which were then visualized with a solution of silver nitrate in phenoxyethanol. GLC allowed the quantitative detection of nanogram amounts of organochlorine insecticides. The electron-capture detector offered high specificity and sensitivity. Good separation of most of the organochlorine pesticides was obtained on pyrex columns, packed with purified, deactivated Chromosorb W (60-80 mesh), coated with 5% Dow 11 silicone grease. The optimum

column temperature was 180-190°C, and purified nitrogen gas flowing at 60-70 ml/min was the mobile phase. Organochlorine pesticides were advantageously extracted from animal tissue by DMF/*n*-hexane partitioning, followed by cleanup on a Florisil column. Chlordane and toxaphene were not adequately separated by this method. Residue determination in fat from Belgians 11 to 71 years old revealed DDE contents of 0.11-3.49 ppm, and DDT contents of 0.10-8.14 ppm.

72-2015. Plato, C. (Div. of Chem. Technol., Food and Drug Adm., Washington, DC 20204). **Differential scanning calorimetry as a general method for determining purity and heat of fusion of high-purity organic chemicals. Application to 64 compounds.** *Anal. Chem. 44(8): 1531-1534; 1972.*

Improvements in a previously published differential scanning calorimetry method for determining chemical purity of pesticides and similar compounds include an initial determination using a fast rate of heating, the use of a faster heating rate for unstable chemicals, and a correction at Δt. The standard deviation of the method was found to be 4% of the impurity measured. The use of an additonal, entirely different method is suggested to confirm purity, since a number of impurities did not depress the melting point. Data for purity determinations of organophosphates, halogenated compounds, carbamates, heterocyclics, and others are included.

72-2016. Greenhalgh, R.; Dokladalova, J.; Haufe, W. O. (Chem. and Biol. Res. Inst., Res. Branch Canada Agr., Ottawa K1A-06C, Canada). **GLC determination of crufomate (Ruelene) in bovine blood and the use of UV irradiation as a confirmatory test.** *Bull. Environ. Contam. Toxicol. 7(4): 237-242; 1972.* (11 references)

Blood samples taken from cattle which had received pour-on treatments with Ruelene were extracted with hexane or chloroform, the extracts being centrifuged to break up the emulsion formed. The solvent was evaporated and the residue redissolved in hexane, then injected directly into the gas chromatograph. A cesium bromide thermionic detector was employed, and the columns were packed with 100/120 Gas Chrom Q coated with 3% OV-17. The columns were preconditioned, and the detector adjusted by varying the hydrogen flow. At a detector temperature of 220°C and a column nitrogen flow of 30 ml/min, the retention time of crufomate was 4.18 min (1.19 relative to parathion). Each sample was bracketed by 1-ng crufomate standard. UV irradiation resulted in the decomposition of all the crufomate after 30 min, leaving a single product with a retention time of 2.8 min. Analysis by GLC/ms showed a parent peak at 257 *m/e* with peaks at 243, 148, 135, 107, and 91 *m/e* and relative abundancies of 100, 62, 60, 19, and 17%. This indicates that the product formed upon irradiation is 4-*tert*-butyl-phenyl methyl methylphosphoramidate and serves as confirmatory evidence for crufomate. Results of determinations at the 0.025-ppm level agreed with bioassay results.

72-2017. Fishbein, L. (Natl. Inst. of Environ. Health Sci., NIH, Pub. Health Serv. and Dept. of Health, Education, and Welfare, Research Triangle Park, NC 27709). **Chromatographic and biological aspects of polychlorinated biphenyls.** *J. Chromatogr. 68(2): 345-426; 1972.* (113 references)

Ecological and toxicological aspects and detailed information concerning analysis of polychlorinated biphenyls (PCB's) are reviewed. The compounds are probably more persistent than DDT and also accumulate in fatty tissue. The most important toxicological effects are long-range sublethal effects which differ markedly between mammals and birds. Column chromatography with Florisil or silicic acid-Celite has been useful in separating PCB's from the organochlorines. Thin-layer chromatography has also been employed. Many examples of thin-layer chromatograms are included. Florisil is also used in gas chromatographic analysis of PCB's; however, an SE-30/QF-1 column usually gives better purification. Results of studies on contaminated wildlife are given which include gas-liquid chromatograms (GLC). Mass spectrometry has been used to enhance GLC methods of identification of PCB's. Using this technique several highly toxic impurities in PCB's, including tetrachlorodibenzofuran, have been identified.

72-2018. Wagenbreth, D.; Trommer, R. (Institut fuer Forstwissenschaften Eberswalde der Deutschen Akademie der Landwirtschaftswissenschaften zu Berlin, DDR). **Eine einfache Biotest-Methode zur Bestimmung der Einwaschung von Herbiziden in Bodensaeulen sowie Moeglichkeiten zur mathematisch-statistischen Auswertung. [A simple bioassay method for determining the leaching of herbicides in soil columns, and suggestions for statistical evaluation of the results.]** *Nachrichtenbl. Pflanzenschutzdienst DDR 25(2): 24-31; 1971.* (12 references) (German)

A simple test setup for studying the leaching of herbicides in soil uses a polyvinyl chloride column 50 cm long and 10 cm in diameter. A panel running along the column for most of its length and covering one-fourth of the circumference is cut out, then held back in place with adhesive tape strips. The column, upended, is filled with soil and supported on filter paper in a Buchner funnel. The pesticide solution is applied uniformly to the top of the column and washed in by spraying on the desired amount of water. The column is left standing until drainage has stopped, then sealed at the top, turned over on its side, and the taped-on panel removed. A plant sensitive to the herbicide tested is sown in transverse rows along the column length. The test plants are observed for the appearance of typical symptoms of herbicide damage at each column band. Suggested statistical evaluation methods are the Wilcoxon test, the long

or short method of testing for extreme values, and the calculation of tolerance limits. Atrazine and simazine leached to 0.5-1.5 cm under these conditions. Prometryne was only detected to a depth of 0.5 cm, possibly due to its low phytotoxicity for the test plants (cress). Potassium chlorate was found in the 16-22 cm soil layer, and dalapon at 22-25 cm, confirming the poor sorbing power of these herbicides despite the high humus content of the soil used. A mixture of proximpham, propham, and diuron produced growth depression only in the row of oats sown at the 1-cm depth, thus indicating that all three components were firmly sorbed in the upper soil layers.

72-2019. Gruebner, P. (Pflanzenschutzamt beim Rat fuer Landwirtschaftliche Produktion und Nahrungsgueterwirtschaft des Bezirkes Dresden, DDR). **Karenzzeitermittlungen fuer Demephion an Gewaechshausgurken. [Waiting-period estimations for Demephion in greenhouse cucumbers.]** *Nachrichtenbl. Pflanzenschutzdienst DDR 25(10): 202-206; 1971.* (27 references) (German)

Demephion residues in treated cucumbers were studied to determine whether the waiting period of 14 days established in East Germany could be reduced to facilitate the use of this insecticide in greenhouse cultures. In addition to the two isomers (PO-demephion and PS-demephion) contained in the parent substance, the analyses also had to detect the sulfoxide and sulfone. The cucumbers were extracted with ethanol, and after evaporation of ethanol, the aqueous residue was saturated with sodium chloride and extracted with chloroform. The chloroform was evaporated and the residue taken up in *n*-hexane. The hexane solution was extracted with water, then the aqueous solution again extracted with chloroform. After evaporation of chloroform, the residue was dissolved in acetone and evaluated semiquantitatively by thin-layer chromatography, using benzene-methanol as the solvent. The method of detection was enzymatic. The residues, determined as the total of the thiol, thiolsulfoxide, and sulfone derivatives, had only decreased to about one-tenth of the initial value within 14 days after treatment, so that the waiting period of 14 days seems necessary. Residues in directly sprayed cucumber fruits and in cucumbers protected with plastic bags during spraying were almost equal after 3 days, showing that the insecticide is introduced to the fruits via the sap.

72-2020. Takahama, K.; Kataoka, S.; Tsunenari, S.; Kakihara, Y.; Ogata, Y.; Kanda, M. (Dept. of Legal Med., Kumamoto U. Med. Sch., Kumamoto, Japan). **Paper-chromatography of synthetic organic pesticides.** *Nippon Hoigaku Zasshi (Jap. J. Leg. Med.) 25(5): 376-382; 1971.* (22 references)

Paper chromatography is often used for pesticide residue detection in forensic medicine because of its simplicity. The identification of 38 organophosphorus, organochlorine, and organomercurial pesticides by paper chromatography was accomplished using a single solvent system and a variety of visualizing agents for each group. Of the 17 organophosphates tested (developer: *n*-butanol:glacial acetic acid:water, 4:1:5 v/v), 12 gave positive reactions with dibromochloroquinonimide (DCQ) reagent, 6 with NaOH reagent, and 15 with Hanes reagent (perchloric acid-ammonium molybdate). Concurrent use of DCQ and Hanes reagents would provide positive identification. All 11 of the organomercurials tested, developed with an *n*-butanol:pyridine:1 *N* ammonia (50:34:16, v/v) solution, gave positive reactions with dithizone, diphenylcarbazone, and diphenylcarbazide. Combined use of dithizone and one of the others is desirable for maximum detectability and specificity. Benzene:ethyl acetate (10:1, v/v) was used as the developer for the 10 organochlorines, and 0.5% tolidine, ammoniacal silver nitrate, or iodine vapor as the visualizing reagents. Positive reactions were obtained with all three reagents, but the first two reagents gave similar *Rf* values for DDT, chlorbenzilate, and dieldrin, as well as for lindane

72-2021. Takahama, K.; Kita, G.; Abe, Y.; Oho, S.; Kawasaki, T.; Kanda, M. (Dept. of Legal Med., Kumamoto U. Med. Sch., Kumamoto, Japan). **Identification of ethylmercuric phosphates by bioautography.** *Nippon Hoigaku Zasshi (Jap. J. Leg. Med.) 25(6): 457-460; 1971.* (5 references)

The usefulness of bioautography in identification of mercurials in tissue has been demonstrated. A rabbit was treated with ethylmercuric phosphate, and tissue samples were taken when the animal died. An ether extract of each tissue was evaporated and the residue taken up in ethanol and chromatographed on paper. The paper was then applied to an agar culture containing mercury-sensitive bacteria, and after a 20-hr incubation the zones of inhibition were measured. A straight-line relationship was obtained with mercury standards of 0.5 to 6.0 μg. Mercury content in various tissues was successfully determined.

72-2022. Takamatsu, C.; Sekida, H.; Takeda, A.; Tanabe, H. (Natl. Inst. for Hyg., Tokyo, Japan). **[Studies on the analysis of pesticide residues in foods. IX. Analytical methods for carbaryl.]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Jap.) 13(3): 244; 1972.* (Japanese)

The methods of determining carbaryl in foods recommended by the Japan Ministry of Agriculture and Forestry (JMAF) and by the AOAC were compared. In the JMAF method, the homogenized vegetable specimen is extracted with methylene chloride; the extract is cleaned up on a moistened alumina column and an

activated carbon-celite column and then treated with a 0.5 *N* methanolic solution of potassium hydroxide and a 0.03% methanolic solution of 4-nitrobenzenediazonium fluoborate. The extinction is read at 580 nm. In the AOAC method, the methylene chloride extract is cleaned up by adding a coagulating reagent, treated with 0.1 *N* methanolic potassium hydroxide and a 0.1% methanolic solution of 4-nitrobenzenediazonium fluoborate (acidified with acetic acid), and the extinction read at 475 nm. Interference by other carbamates is greater in the case of the JMAF method, but the color stability with this method is better. However, the use of an additonal activated carbon column for cleanup in the AOAC method is recommended to remove excessive vegetable pigments. A combination of the two methods may prove optimal.

72-2023. Torii, I.; Matsunaga, K.; Ogino, Y.; Morita, K.; Narimoto, T.; Soga, T. (Okayama Prefectural Inst. for Hyg., Okayama-shi, Japan). **[Analytical studies for detecting minute constituents in foods. IV. Quantitative analytical separation method for pesticide residues by dialysis (I).]** *Shokuhin Eiseigaku Zasshi (J. Food Hyg. Soc. Jap.) 13(3): 246; 1972.* (Japanese)

An improved method for separating organochlorine pesticide residues from milk fat by dialysis is described. Two grams of specimen, 10 ml of *n*-hexane, and 8 ml of acetonitrile were placed in a cellulose tube; and the tube was immersed in 160 ml of acetonitrile for 25 hr at room temperature with stirring. Then the dialysis fluid was extracted with 700 ml of 2% aqueous sodium chloride solution and 100 ml *n*-hexane. The hexane was evaporated, and the concentrated extract analyzed by gas chromatography with an electron-capture detector. The results were compared with those obtained with the modified FDA method. A large peak was found at $Rf = 0.43$, which interfered with the determination of lindane and could not be removed by passing the extract over a Florisil column. Excessive values for lindane and dieldrin compared to those found with the FDA method are apparently due to overlapping with impurity peaks.

72-2024. Torii, I.; Matsunaga, K.; Ogino, Y.; Morita, K.; Narimoto, T.; Soga, T. (Okayama Prefectural Inst. for Hyg., Okayama-shi, Japan). **[Analytical studies for detecting minute constituents in foods. V. Quantitative analytical separation method for pesticide residues by dialysis (II).]** *Shokuhin Eiseigaku Zasshi (J. Foód Hyg. Soc. Jap.) 13(3): 246-247; 1972.* (Japanese)

To permit direct extraction of organochlorine pesticide residues from milk specimens without prior separation of milk lipids, the dialysis method described in the previous report (Abstract No. 72-2023) was modified. A cellulose dialysis tube was preconditioned by immersing in acetonitrile-4% aqueous acetic acid at room temperature for 2 days. After rinsing, the tube was filled with 10 g of milk, 6 ml of *n*-hexane, and 4 ml of acetonitrile. It was then immersed in 180 ml of acetonitrile for 25 hr at room temperature. The entire volume of dialysis fluid was then extracted with saline solution/*n*-hexane as described in part I and subjected to gas chromatography. Good recoveries of BHC isomers, dieldrin, and DDT and its derivatives were obtained, without the large interfering peaks obtained previously. Various other methods of modifying the dialysis tube were tried, such as immersion in acetonitrile-hydrochloric acid or immersion in acetonitrile-acetic acid of different concentrations. However, the method described gave the best results.

72-2025. Guth, J. A.; Voss, G. (Agrochem. Div., Ciba-Geigy Ltd., Basel, Switzerland). **Automated colorimetric procedure for the determination of total and unchanged urea herbicide residues in soil.** *Weed Res. 11(2/3): 111-119; 1971.* (18 references)

An improved procedure for determination of urea herbicide residues is suitable for the routine analysis of large numbers of soil samples, saving time and labor. For total residue determination (unchanged herbicide plus metabolites retaining the aniline structure), the soil samples are subjected to combined alkaline hydrolysis, distillation, and extraction into 2,2,4-trimethylpentane in the Bleidner apparatus. For determination of only the remaining unchanged compound, soil samples are Soxhlet-extracted with acetone-water-HCl, and after acetone evaporation, the dichloromethane extract of the residue purified by TLC. Preferred developers are chloroform:ethanol (95:5) for metobromuron, chlorbromuron, and chlortoluron, or dichloromethane:ethanol (95:5) for fluometuron. The alumina scraped off from the marked spots, which contain the herbicide, is then subjected to the same alkaline hydrolysis-distillation-extraction as before. The respective trimethylpentane extracts are then extracted with 1 *N* HCl, and the resulting solutions analyzed colorimetrically by an automated method. The flow diagram for the automated determination differs from previously described manifolds by the insertion of a debubbler for nitrogen removal after addition of sulfamic acid, the use of *N*-ethyl-1-naphthylamine as the coupling reagent for the diazotized anilines, and the addition of heating baths for maximum color development. Recoveries of 87-114% were obtained at the 0.2-5.0 ppm residue level.

72-2026. Horowitz, M.; Hulin, N. (Div. of Weed Res., Volcani Inst. of Agr. Res., Newe Ya'ar Exper. Sta., P.O. Haifa, Israel). **A rapid bioassay for diphenamid and its application in soil studies.** *Weed Res. 11(2/3): 143-149; 1971.* (10 references)

For bioassay of diphenamid in soil, petri dishes were filled with an untreated substrate on which an aqueous diphenamid solution was pipetted, or with a moistened soil-herbicide mixture. Ten pregerminated oat

seeds were placed in a row on a filter pad in the center of the dish; then the covered dish incubated in the dark at 30°C for 24 hr. After this incubation, the dishes were removed from the incubator, and the length of the longest root of each seed was marked with wax pencil on the lid of the dish. The dishes were quickly returned to the incubator and incubated for another 24 hr, and the root lengths were again measured. Progressive reduction of root length with increasing diphenamid concentration was evident after 24 hr of incubation and was more striking after 48 hr. Studies on the influence of soil characteristics on herbicide bioactivity indicated that the organic matter content of the soils had the greatest influence on adsorption-release phenomena. The good correlations between initial and recovered activity in adsorption-release experiments showed that diphenamid-soil studies can be simplified by using the rapid indirect method, employing the determination of herbicide bioactivity in aqueous soil extracts applied to inert substrates.

72-2027. Scifres, C. J.; Bovey, R. W.; Merkle, M. G. (Dept. of Range Sci., ARS, U.S. Dept. of Agr., Texas A & M U., College Station, TX). **Variation in bioassay attributes as quantitative indices of picloram in soils.** *Weed Res. 12(1): 58-64; 1972.* (19 references)

Picloram contained in nanogram quantities in river sand was more readily quantified with the aid of phytotoxicity symptom ratings than using variations in fresh or oven-dry weights of indicator plants. A chart of ratings corresponding to specific symptoms in the test plants was prepared. When log-dose versus symptom-rating lines were calculated, 95% of the variation in response of Black Valentine beans could be attributed to variation in picloram level. Symptom ratings of Black Valentine beans were accurate in estimating 4 ppb of picloram in sand, but usually underestimated higher concentrations. Soybean symptom ratings slightly underestimated the low concentrations and overestimated the 250-ppb concentration. Sunflower estimates were similar to those with soybeans, with increased error at the higher concentrations. Cucumbers were insensitive to picloram below 10 ppb and thus gave a biased concentration curve. Results from bioassays with Black Valentine beans and soybeans were only slightly more variable than GLC analyses and thus recommend themselves as a simple, reliable method of quantitative analysis.

72-2028. Tanabe, H. (Food Dept., Natl. Hyg. Lab., Japan). **[Analysis of PCB in food.]** *Jap. Food Sci. (Jap. Fudo Saiensu) 11(5): 85-92; 1972.* (Japanese)

A method for determination of polychlorinated biphenyls (PCB) in food is presented. A PCB mixture may contain more than 200 chlorinated biphenyls; 50 of these have been separated using a special gas chromatographic column. Sample workup begins with alkaline hydrolysis of a concentrated *n*-hexane extract of a mixture containing PCB and other halogenated hydrocarbons and then reextraction with *n*-hexane. The extract is further cleaned up on a silica gel column. The concentrate, containing PCB and DDE, is then subjected to gas chromatography with an electron-capture detector. For separation of PCB from chlorinated insecticides, the sample is first partitioned in acetonitrile/*n*-hexane, then washed repeatedly with water. The organic layer is purified on a chromatographic column. Both qualitative and quantitative determinations of PCB are performed by gas chromatography with an electron-capture detector.

72-2029. Willis, D. E.; Addison, R. F. (Fisheries Res. Bd. of Canada, Marine Ecol. Lab., Bedford Inst., Dartmouth, N.S., Canada). **Identification and estimation of the major components of a commercial polychlorinated biphenyl mixture, Aroclor 1221.** *J. Fish. Res. Bd. Can. 29(5): 592-595; 1972.* (13 references)

The quantitative distribution of components in Aroclor 1221 was determined by synthesis of presumed components and comparison of their TLC and GLC behavior with that of the sample. Retention times on both polar and nonpolar columns of a GLC instrument with an electron-capture detector were employed in assignment of peaks to synthesized compounds. Quantitation indicated the presence of 12.7% biphenyl; 28.4% 2-chlorobiphenyl; 18.7% 4-chlorobiphenyl; 9.2% 2,2′-dichlorobiphenyl; 3.5% 2,4-dichlorophenyl; 13.6% 2,4′-dichlorobiphenyl; and 6.2% 4,4′-dichlorobiphenyl. This composition accounted for 92.3% of the Aroclor 1221 sample, and the calculated wt-% of Cl in this composition was 19.6%. Several late-emerging chromatographic peaks might have been due to trichlorobiphenyls, but could not be identified with certainty.

72-2030. Miyachi, Y.; Shoyama, M. (Criminol. Lab., Aichi Prefectural Police Headquarters, Nagoya-shi, Japan). **[Gas chromatographic analysis of constituent phenols of *N*-methylcarbamate insecticides.]** *Kagaku Keisatsu Kenkyusho Hokoku (Rep. Nat. Res. Inst. Police Sci.) 25(1): 29-35; 1972.* (11 references) (Japanese)

For qualitative and quantitative analysis of commercial *N*-methylcarbamate insecticides, the technical or formulated materials were first cleaned up by recrystallization from acetone until only a single TLC spot was obtained for each compound. The carbamates investigated were Hopcide (CPMC), Suncide (propoxur), Tsumacide (MTMC), Mipcine (*m*-isopropylphenyl *N*-methylcarbamate), Cosban (3,5-xylenyl *N*-methylcarbamate), Bassa (BMPC), meobal, and Denapon (carbaryl). The purified specimens were then hydrolyzed with 0.5 *N* methanolic KOH, and the resulting phenol of α-naphthol extracted with ether and adsorbed on a silica gel column. After developing with petroleum ether, the samples were eluted with benzene, and the benzene solutions injected into the gas chromatograph. A GLC

column containing 2.5% Apiezon L on Diasolid L afforded separation of all eight types of insecticide, while that containing 12% Apiezon L on celite 545 separated only seven. The former column was operated at 135-210°C (programmed at 10°C/min); 10 min were allowed after injection; and Apiezon-celite column was operated isothermally at 178°C. The operating conditions were: carrier gas, He at 25 ml/min; flame ionization detector; injection temperature, 250°C.

72-2031. Bazzi, B.; Abbruzzese, L.; Galluzzi, G.; Nesti, V. (Ricerche e Brevetti, Istituto di Ricerche Agrarie, Montecatini Edison S.p.A., Milan, Italy). **Gas-chromatographic determination of the residues of 2-fluoroethyl-4-biphenylacetate (fluenethyl) in/on apples and pears.** *Pestic. Sci. 2(6): 251-254; 1971.* (11 references)

A specific method for the determination of fluenethyl in apples, pears, and peaches is described. The pesticide was extracted from the fruit with acetone and the extract purified by means of several partitions with *n*-hexane and acetonitrile and by silica gel column chromatography. The material was brominated and again chromatographed on silica gel. Brominated samples were analyzed by GLC using an electron-capture detector with 10 mCi of ^{90}Sr as the radioactive source. The limit of sensitivity was 0.0025 ppm, and no metabolites were found to interfere with the fluenethyl assay.

72-2032. Crofts, M.; Harris, R. J.; Whiteoak, R. J. (Fisons Ltd., Agrochem. Div., Chesterford Park Res. Sta., Nr. Saffron Walden, Essex, England). **Determination of residues in apples and pears following treatment with the acaricide fenazaflor. I. Routine method for fenazaflor and major metabolites.** *Pestic. Sci. 3(1): 29-37; 1972.* (6 references)

A method is described for the determination of residues of fenazaflor (I), its hydrolysis product 5,6-dichloro-2-trifluoromethylbenzimidazole (II), and the metabolite 5,6-dichloro-4-hydroxy-2-trifluoromethylbenzimidazole (III) in apples and pears. These chemicals may be present in the crop as a result of the application of the acaricide fenazaflor, formulated as a wettable powder, and known by the trade name 'Lovozal.' Total residues of I and II are determined as II, and III is determined after methylation, both by GLC with electron-capture detection. The limit of detection for each compound is 0.02 ppm (based on a 50-g sample weight). Recoveries are over 80%. Using apples sprayed with radio-labeled fenazaflor it was shown that approximately 20% of the total radioactivity present in the fruit was not extracted by the routine method. Further investigation showed much of this to be present as trifluoroacetic acid in a tightly bound form. No problems were encountered with interference from a number of commonly used orchard pesticides. (Author abstract by permission)

72-2033. Crofts, M.; Harris, R. J.; Whiteoak, R. J. (Fisons Ltd., Agrochem. Div., Chesterford Park Res. Sta., Nr. Saffron Walden, Essex, England). **Determination of residues in apples and pears following treatment with the acaricide fenazaflor. II. Method for individual residues of fenazaflor and its hydrolysis product.** *Pestic. Sci. 3(1): 39-42; 1972.* (1 reference)

The occasional need for separation of residues of fenazaflor and its hydrolysis product, instead of determination of the total residue by the routine method, is satisfied by partition of the extract between hexane and acidified aqueous methanol. Both residues are subsequently determined as the hydrolysis product by GLC with electron-capture detection, after hydrolysis of fenazaflor. (Author abstract by permission)

72-2034. Kolbezen, M. J.; Abu-El-Haj, F. J. (U. of California, Riverside, CA 92502). **Fumigation with methyl bromide. II. Equipment and methods for sampling and analysing deep field soil atmospheres.** *Pestic. Sci. 3(1): 73-80; 1972.* (1 reference)

To determine the concentrations of methyl bromide fumigant in soil, a series of sampling stations was placed at defined distances from the fumigant charge. Each sampling station consisted of five stainless steel tubes fitted into a piece of channel iron and held in place by an aluminum cover. Each tube had a brass screen at its lower end to prevent clogging of the tip with soil and a rubber septum arrangement at its upper end, suitable for piercing by the sampling syringe. A hole of the desired depth was dug by soil auger and the sampling assembly inserted. About 5-7 cm of sand was poured around each sampling point, and the intervening spaces tightly packed with soil. Samples were taken in syringes lubricated and sealed with a nonionic surfactant. The samples, transported to the laboratory, were analyzed by gas chromatography, using an appropriate set of methyl bromide standards. It was not necessary to make any allowances in the calculations for sample loss during transport or for adsorption on tubing or syringes; both were negligible within the experimental accuracy. Reliable results (within 10%) were obtained between 10 ppm and 100 parts per thousand.

72-2035. Thimm, H. F.; Belcher, R. S.; Briner, G. P. (Dept. of Agr., Victoria, 5 Parliament Pl., Melbourne, Victoria 3002, Australia). **Estimation of dieldrin in cured tobacco.** *Pestic Sci. 3(2): 175-177; 1972.* (6 references)

Previous methods for the determination of organochlorine insecticides in tobacco, employing gas-liquid chromatography with an electron-capture detector, have suffered from the excessively high artifact levels appearing in the chromatogram. In an improved method, tobacco leaf was blender extracted with pentane, and the sample volume reduced in a Kuderna-Danish evaporator. Triplicate Storherr tubes were packed with

layers of glass beads and sand, bounded by glass wool, and equilibrated in the sweep-codistillation instrument using nitrogen as the carrier gas. The Storherr tubes were flushed with ethyl acetate, then 2 ml of the extract in hexane injected, followed by further 2-ml injections of hexane at 2-min intervals for 15 min. The collected and concentrated solution was chromatographed on a TLC plate with silver nitrate-impregnated alumina. The alumina from the sample front at an *Rf* equal to that of the standard spot was scraped off and extracted 10 times with ethyl acetate. Finally, this extract was concentrated and chromatographed on a column containing equal parts of 10% DC-200 and 15% QF-1 on Gas Chrom Q support. Recoveries of 98-100% were obtained for dieldrin from tobacco leaf spiked at 0.8 ppm. The detection level was 0.01 ppm. The interference peaks found by previous methods were not noted.

72-2036. Williams, I. H. (Res. Sta., Canada Dept. of Agr., 6600 N.W. Marine Dr., Vancouver 8, B.C., Canada). **Direct gas chromatographic determination of methomyl (Lannate) with microcoulometric nitrogen detection.** *Pestic. Sci. 3(2): 179-181; 1972.* (5 references)

A microcoulometric method is described for the direct determination of nanogram amounts of methomyl (*S*-methyl-*N*-[(methylcarbamoyl)-oxy] thioacetimidate). An all-glass gas chromatographic system coupled to a microcoulometer with a nitrogen-specific cell was used. The column was packed with a mixture of 6% OV210 and 4% 0V101 on Gas Chrom Q, as was the glass transfer tube which connected the chromatographic column to the pyrolysis furnace. Some loss occurred but response was linear from 125 to 20 ng, and sensitivity was such that 20 ng of methomyl could be readily measured. (Author abstract by permission)

72-2037. Burkhard, N.; Voss, G. (CIBA-GEIGY Ltd., Agrochem. Div., Basel, Switzerland). **Gas chromatographic residue method for iodofenphos and two of its metabolites in animal tissues, fat and milk.** *Pestic. Sci. 3(2): 183-190; 1972.* (6 references)

A gas chromatographic residue method for use on animal tissues, fat, and milk is presented for the organophosphorus insecticide iodofenphos. The procedure includes the determination of two potential metabolites, iodofenoxon, and 2,5-dichloro-4-iodophenol. The two phosphorus-containing compounds are analyzed by means of a flame photometric detector after cleanup including silica gel column and thin-layer chromatography. The substituted phenol is determined separately as the acetylated derivative using an electron-capture detector. Some information on the problem of specificity with regard to possible interferences by closely related insecticides or their corresponding phenols is also given. (Author abstract by permission)

See also **72-1866** **72-1954** **72-1967** **72-1981**

HAPAB QUARTERLY INDEX

JULY–SEPTEMBER 1972, Vol. 5, Nos. 7-9

PREFACE

The basic design for this index was supplied by the Division of Pesticide Community Studies. It consists of two author indexes (personal and corporate) and two subject indexes (compound and concept).

Compounds are indexed by their common names; where other compound designations are used by authors, these synonyms appear with the notation to *see* the common name. Articles referring, in general, to structural or functional chemical classes are indexed under these broader headings (e.g., organochlorines, herbicides, etc.).

Subheadings in the compound index are identical with the main headings in the concept index, the latter having a maximum of three levels of specificity in addition to the compound.

Following is a cross-referenced guide to the subject headings:

Alternative Controls
Analysis
Antidotes (*see* Treatment of Poisoning)
Associations/Organizations
Beneficial Effects
Biochemical Effects (*see also* Enzyme Activity)
Blood/Body Fluids
Carcinogenesis
Cardiovascular System
Cholinesterase (*see* Enzyme Activity)
Cholinesterase-Measurement (*see* Analysis - Cholinesterase/Enzyme)
Chromatography (*see* Analysis - Chromatography)
Conferences
Cytological Effects
Decontamination (*see* Residue Removal)
Degradation (*see* Residue Degradation *and* Metabolism)
Dermal Effects (*see* Integument)
Diet (*see* Factors Influencing Metabolism/Toxicity - Nutritional State)
Digestive System
Domestic Animals (*see* Toxicity/Domestic Animals *and* Residues/Domestic Animals)
Economics
Education/Training (*see also* Prevention)
Embryology (*see* Reproduction/Growth - Embryo/Fetus)
Endocrine System (*see also* Reproduction/Growth)
Environmental Pollution
Enzyme Activity (*see also* Biochemical Effects)
Excretory System
Experimental Animals (*see* Toxicity/Experimental Animals)
Experimental Design
Factors Influencing Metabolism/Toxicity
Food and Feed (*see* Residues/Food and Feed)
Human exposure (*see* Toxicity/Humans *and* Residues/Humans)
Immunology
Integument
Interactions (*see* Factors Influencing Metabolism/Toxicity)
Laws and Regulations (*see also* Prevention)
LD50 (*see* Toxicity/Experimental Animals)
Metabolism (*see also* Residue Degradation)
Microscopy, light and electron (*see* Cytological Effects)
Morbidity and Mortality Statistics
Musculoskeletal System
Mutagenesis/Teratogenesis (*see also* Reproduction/Growth)
Nervous System
Nomenclature
Photodecomposition (*see also* Residue Degradation)
Plants (*see* Residues/Plants)
Prevention (*see also* Laws and Regulations *and* Education/Training)
Reproduction/Growth (*see also* Endocrine System *and* Mutagenesis/Teratogenesis)
Residue Degradation (*see also* Photodecomposition *and* Metabolism)
Residue Dynamics
Residue Removal
Residues/Air
Residues/Domestic Animals
Residues/Food and Feed
Residues/Human
Residues/Plants (other than food and feed)
Residues/Soil
Residues/Water
Residues/Wildlife
Respiratory System
Reviews
Sensory System
Skeleton (*see* Musculoskeletal System)
Soil (*see* Residues/Soil)
Teratogenesis (*see* Mutagenesis/Teratogenesis)
Toxicity/Domestic Animals
Toxicity/Experimental Animals
Toxicity/Humans
Toxicity/Wildlife
Therapeutic Use
Treatment of Poisoning
Water (*see* Residues/Water)
Wildlife (*see* Residues/Wildlife)

Agallol
see also Fungicides, Mercurials
Residues/Soil 72-1389
Residues/Water 72-1389
Alachlor
see also Herbicides
Residue Degradation 72-1414
Aldrin
see also Organochlorines
Analysis 72-1553 72-1781 72-1793 72-1810
Beneficial Effects 72-1367
Biochemical Effects 72-1521 72-1878
Digestive System 72-1878
Environmental Pollution 72-1367
Enzyme activity 72-1480 72-1484 72-1521 72-1878
Factors Influencing Metabolism/Toxicity 72-1963
Metabolism 72-1401 72-1405 72-1408 72-1431 72-1908 72-1935 72-1962 72-1963
Photodecomposition 72-1842
Residue Degradation 72-1618 72-1842
Residue Dynamics 72-1629
Residue/Air 72-1642
Residues/Domestic Animals 72-1618
Residues/Food and Feed 72-1395 72-1401 72-1405 72-1408 72-1618 72-1643 72-1832
Residues/Humans 72-1395 72-1660 72-1878
Residues/Plants 72-1403 72-1404 72-1431
Residues/Water 72-1643 72-1781
Toxicity/Experimental Animals 72-1480 72-1492 72-1521 72-1726 72-1908
Toxicity/Humans 72-1878
Toxicity/Wildlife 72-1367
Allethrin
see also Botanicals
Blood/Body Fluids 72-1933
Toxicity/Experimental Animals 72-1933
Alodan
see Chlorbicyclen
Aluminum phosphide
see also Fumigants
Analysis 72-1624
Residue Removal 72-1388
Residues/Food and Feed 72-1388
Ametryne
see also Herbicides
Residues/Soil 72-1855
4-Amino-6-tert-butyl-3-(methylthio)-1,2,4-triazin-5-one
see BAY 94337
Amiton
see also Organophosphates
Enzyme activity 72-1511
Amitril
see Amitrole
see Ammonium Thiocyanate
Amitrole
see also Herbicides
Analysis 72-2012
Education/Training 72-1354
Factors Influencing Metabolism/Toxicity 72-1720
Integument 72-1720
Laws and Regulations 72-1354
Musculoskeletal System 72-1720
Mutagenesis/Teratogenesis 72-1720
Prevention 72-1354
Toxicity/Experimental Animals 72-1720
Ammonium Thiocyanate
see also Herbicides
Education/Training 72-1354
Laws and Regulations 72-1354
Prevention 72-1354
Amo 1618
see also Herbicides
Biochemical Effects 72-1764
Toxicity/Experimental Animals 72-1764
Anthio
see Formothion
Antidotes
see also Pralidoxime
Antio
see Formothion
Apholate
see also Chemosterilants
Mutagenesis/Teratogenesis 72-1971
Reviews 72-1971
Aroclor 1242
see Polychlorinated biphenyls
Aroclor 1254
see Polychlorinated biphenyls
Arsenicals
see also Herbicides, Sodium arsenite, TTCA
Analysis 72-1576
Environmental Pollution 72-1606
Metabolism 72-1400
Residues/Food and Feed 72-1400 72-1576 72-1852
Reviews 72-1602 72-1606
Atrazine
see also Herbicides
Metabolism 72-1506 72-1956
Residue Degradation 72-1850
Residue Dynamics 72-1850
Residues/Soil 72-1375 72-1854 72-1855
Avadex
see Di-allate
Avicides
see also 3-Chloro-p-toluidine, Fenthion
Azinphosmethyl
see also Organophosphates
Analysis 72-1794 72-2006

Enzyme activity
72-1485
Factors Influencing Metabolism/Toxicity
72-1485
Metabolism
72-1485
Photodecomposition
72-1647
Residue Degradation
72-1433 72-1647
Residues/Food and Feed
72-1433
Toxicity/Experimental Animals
72-1485

Bandane
see also Herbicides
Biochemical Effects
72-1521
Enzyme activity
72-1521
Toxicity/Experimental Animals
72-1521

Barban
see also Carbamates, Herbicides
Digestive System
72-1474
Metabolism
72-1752
Prevention
72-1452
Residue Degradation
72-1752
Toxicity/Experimental Animals
72-1474
Toxicity/Humans
72-1452

Baygon
see Propoxor

BAY 6159 H
see BAY 94337

BAY 94337
Toxicity/Experimental Animals
72-1748

Benefin
see also Herbicides, herbicides
Analysis
72-1580
Residues/Soil
72-1580 72-1663

Benomyl
see also Fungicides
Factors Influencing Metabolism/Toxicity
72-1889
Immunology
72-1889
Integument
72-1889
Residues/Food and Feed
72-1411 72-1863
Toxicity/Humans
72-1889

Bensulide
see also Herbicides
Mutagenesis/ Teratogenesis
72-1510
Residues/Soil
72-1663

BHC
see also Organochlorines
Analysis
72-1583
Digestive System
72-1486
Excretory System
72-1486
Factors Influencing Metabolism/Toxicity
72-1759
Immunology
72-1771
Metabolism
72-1759
Prevention
72-1448
Reproduction/Growth
72-1486
Residue Dynamics
72-1638
Residue Removal
72-1638
Residues/Food and Feed
72-1832 72-1835
72-1836
Residues/Humans
72-1380 72-1390
72-1660
Residues/Soil
72-1638
Residues/Water
72-1638
Toxicity/Experimental Animals
72-1486
Toxicity/Humans
72-1380

beta-BHC
see BHC isomers

BHC isomers
Analysis
72-1552 72-1553
Factors Influencing Metabolism/Toxicity
72-1388
Metabolism
72-1401 72-1405
72-1773 72-1987
72-1988 72-1989
Residues/Food and Feed
72-1401 72-1402
72-1405 72-1406
72-1643
Residues/Humans
72-1644
Residues/Plants
72-1403 72-1404
Residues/Soil
72-1407
Residues/Water
72-1407 72-1643

Biphenyl
see also Fungicides
Enzyme activity
72-1738
Factors Influencing Metabolism/Toxicity
72-1738
Metabolism
72-1738

2,2-Bis(p-chlorophenyl)-1,1-dichloroethane
see TDE

Botanicals
see also Allethrin, Pyrethrins, Rotenone

Bromacil
see also Herbicides
Analysis
72-1562

Bromoxynil
see also Herbicides
Analysis
72-1398 72-1562
Residue Degradation
72-1398

O-n-Butyl-S-n-butyl-methylthiophosphonate
see GA-3

Calcium cyanamide
Reproduction/Growth
72-1927

Captafol
see also Fungicides
Integument
72-1684 72-1686
Morbidity and Mortality Statistics
72-1686
Toxicity/Humans
72-1684

Captan
see also Fungicides
Environmental Pollution
72-1355
Factors Influencing Metabolism/Toxicity
72-1513
Mutagenesis/ Teratogenesis
72-1890
Respiratory System
72-1513
Toxicity/Experimental

Animals
72-1513
Toxicity/Humans
72-1355
Carbamates
see also Barban, Carbaryl, Di-allate, Dicresyl, m-(N,N-Dimethylformamidine) phenyl N-methylcarbamate HCl, EPTC, Landrin, Methiocarb, Methomyl, Mobam, Molinate, NH-6367, NH-6967, Vanalate, Zineb
Analysis
72-1547 72-1578
72-1635 72-1797
72-2015 72-2030
Environmental Pollution
72-1843
Enzyme activity
72-1499
Metabolism
72-1923 72-1948
72-1958 72-1965
Residue Degradation
72-1635 72-1843
Residues/Soil
72-1547
Reviews
72-1578 72-1843
72-1948 72-1958
Sensory System
72-1695
Toxicity/Domestic Animals
72-1695 72-1894
Toxicity/Experimental Animals
72-1518
Carbaryl
see also Carbamates
Analysis
72-1550 72-1564
72-1568 72-1570
72-1981 72-2022
Blood/Body Fluids
72-1964
Enzyme activity
72-1527 72-1964
Factors Influencing Metabolism/Toxicity
72-1496 72-1513
72-1964
Metabolism
72-1478 72-1507
72-1541 72-1935
72-1964 72-1981
72-1997
Mutagenesis/ Teratogenesis
72-1993
Reproduction/Growth
72-1964 72-1986
72-1993
Residue Degradation
72-1478
Residues/Plants
72-1564
Respiratory System
72-1513
Toxicity/Experimental Animals
72-1496 72-1513
72-1527 72-1541
72-1749 72-1964
72-1986 72-1993
Carbetox
see Malathion
Carbofenthion
Residue Degradation
72-1849
Carbyne
see Barban
CCC
see Chlormequat chloride
Celphos
see Aluminum phosphide
Ceresan L
Metabolism
72-1473
Nervous System
72-1473
Toxicity/Experimental Animals
72-1473
Chemosterilants
see also Apholate, Hempa
Environmental Pollution
72-1355
Toxicity/Humans
72-1355
Chlodithane
see TDE
Chloramphenicol
Analysis
72-1804
Chlorbicyclen
see also Organochlorines
Metabolism
72-1530
Chlorbromuron
see also Herbicides
Analysis
72-2025
Chlordane
see also Organochlorines
Biochemical Effects
72-1521 72-1522
72-1523
Enzyme activity
72-1521 72-1522
72-1523
Factors Influencing Metabolism/Toxicity
72-1708
Metabolism
72-1708 72-1715
Reproduction/Growth
72-1598
Residues/Plants
72-1651
Residues/Soil
72-1651
Residues/Water
72-1385
Reviews
72-1598
Toxicity/Experimental Animals
72-1521 72-1522
72-1523 72-1708
Toxicity/Wildlife
72-1598
Chlordecone
see also Organochlorines
Analysis
72-1560
Chlordimeform
Residue Degradation
72-1421 72-1422
Residues/Food and Feed
72-1421 72-1422
Chlorinated naphthalenes
Analysis
72-1561
Chlormequat chloride
see also Herbicides
Biochemical Effects
72-1768
Digestive System
72-1768
Enzyme activity
72-1768
Mutagenesis/ Teratogenesis
72-1971
Reproduction/Growth
72-1768
Reviews
72-1971
Toxicity/Experimental Animals
72-1768
2-Chloro-2',6'-diethyl-N-(methoxymethyl) acetanilide
see Alachlor
2-(Chloroethyl) phosphonic acid
see Ethephon
2-Chloroethylphosphonic acid
see Ethephon
Chlorofos
see Trichlorfon
S-(p-Chloro-alpha-phenylbenzyl) O,O-diethyl phosphorodithioate
see R-3828
Chlorophos
see Trichlorfon

Chloropicrin
see also Fumigants
Factors Influencing
Metabolism/Toxicity
72-1458
Prevention
72-1458
Toxicity/Humans
72-1458
3-Chloro-p-toluidine
see also Avicides
Enzyme activity
72-1979
Factors Influencing
Metabolism/Toxicity
72-1979
Metabolism
72-1979
2-Chloro-1-(2,4,5-trichlorophenyl) vinyl dimethyl phosphate
see Tetrachlorvinphos
Chlorpropham
see also Herbicides
Factors Influencing
Metabolism/Toxicity
72-1513
Metabolism
72-1723
Respiratory System
72-1513
Toxicity/Experimental Animals
72-1513
Chlorthiamid
see also Herbicides
Analysis
72-1399
Residue Degradation
72-1399
Residues/Food and Feed
72-1399
Chrysanthemic acid
see Pyrethrins
Chwastoks
see 2,4-D derived compounds
CIPC
see Chlorpropham
Copper sulfate
see also Fungicides
Digestive System
72-1543
Toxicity/Experimental Animals
72-1543
Co-Ral
see Coumaphos
Cotoran
see Fluometuron
Coumaphos
see also Organophosphates
Enzyme activity
72-1485
Factors Influencing
Metabolism/Toxicity
72-1485
Metabolism
72-1485
Residue Degradation
72-1849
Toxicity/Experimental Animals
72-1485
Crufomate
see also Organophosphates
Analysis
72-2016
O-p-Cyanophenyl O-ethyl phenylphosphonothioate
see S 4087
Cygon
see Dimethoate
C-2307
see also Organophosphates
Metabolism
72-1753

2,4-D
see also Herbicides
Analysis
72-1569
Biochemical Effects
72-1768
Cytological Effects
72-1534
Digestive System
72-1768 72-1950
Education/Training
72-1354
Enzyme activity
72-1768 72-1973
Laws and Regulations
72-1354
Metabolism
72-1955 72-2001
Musculoskeletal System
72-1995
Mutagenesis/Teratogenesis
72-1534 72-1995
Nervous System
72-1950
Prevention
72-1354
Reproduction/Growth
72-1534 72-1768
72-1973 72-1991
Residue/Air
72-1833
Residues/Food and Feed
72-1655
Toxicity/Experimental Animals
72-1768 72-1950
72-1991 72-1995
2,4-D derived compounds
Enzyme activity
72-1973
Reproduction/Growth
72-1973
Dalapon
see also Herbicides
Education/Training
72-1354
Laws and Regulations
72-1354
Prevention
72-1354
Dasanit
see Fensulfothion
2,4-DP
Metabolism
72-2001
DDA
see DDT derived compounds
DDD
see TDE
o,p'-DDD
see TDE
DDE
see DDT derived compounds
o,p'-DDE
see DDT derived compounds
p,p'-DDE
see DDT derived compounds
DDMU
see DDT derived compounds
DDT
see also Organochlorines
Alternative Controls
72-1364
Analysis
72-1548 72-1575
72-1780 72-1781
72-1784 72-1791
72-1792 72-2013
Beneficial Effects
72-1367 72-1512
72-1584
Biochemical Effects
72-1471 72-1521
72-1696 72-1751
72-1775 72-1891
72-1910 72-1915
72-1916
Blood/Body Fluids
72-1696 72-1710
72-1757 72-1891
72-1964
Carcinogenesis
72-1364 72-1481
72-1926
Cardiovascular System
72-1696 72-1710

Cytological Effects
72-1475 72-1501
72-1916
Digestive System
72-1475 72-1493
72-1526 72-1543
72-1732 72-1757
72-1758 72-1910
Endocrine System
72-1758 72-1942
Environmental Pollution
72-1355 72-1357
72-1361 72-1364
72-1367 72-1373
72-1584 72-1594
72-1603
Enzyme activity
72-1465 72-1481
72-1493 72-1500
72-1501 72-1521
72-1526 72-1527
72-1751 72-1757
72-1891 72-1910
72-1916 72-1964
Excretory System
72-1732 72-1910
Experimental Design
72-1357
Factors Influencing Metabolism/Toxicity
72-1435 72-1475
72-1493 72-1496
72-1513 72-1526
72-1615 72-1645
72-1711 72-1713
72-1714 72-1741
72-1755 72-1772
72-1845 72-1907
72-1910 72-1912
72-1963 72-1964
Integument
72-1449
Laws and Regulations
72-1359 72-1823
72-1826
Metabolism
72-1405 72-1475
72-1487 72-1489
72-1711 72-1713
72-1714 72-1739
72-1741 72-1755
72-1756 72-1772
72-1776 72-1845
72-1848 72-1853
72-1873 72-1908
72-1912 72-1915
72-1941 72-1948
72-1962 72-1963
72-1964 72-1982
Morbidity and Mortality Statistics
72-1891
Mutagenesis/ Teratogenesis
72-1449
Nervous System
72-1471 72-1758
72-1775 72-1907
Prevention
72-1448
Reproduction/Growth
72-1438 72-1449
72-1483 72-1512
72-1533 72-1598
72-1658 72-1659
72-1669 72-1710
72-1741 72-1757
72-1758 72-1939
72-1940 72-1964
Residue Degradation
72-1386 72-1425
72-1776 72-1791
72-1868 72-1873
72-1982
Residue Dynamics
72-1425 72-1756
Residue Removal
72-1839
Residue/Air
72-1357 72-1429
72-1620 72-1833
Residues/Food and Feed
72-1395 72-1402
72-1405 72-1420
72-1548 72-1627
72-1643 72-1661
72-1666 72-1667
72-1710 72-1792
72-1832 72-1839
72-1848 72-1851
Residues/Humans
72-1378 72-1380
72-1390 72-1395
72-1435 72-1615
72-1637 72-1660
72-1696 72-1876
72-1891
Residues/Plants
72-1386 72-1403
72-1404 72-1623
72-2013
Residues/Soil
72-1620 72-1636
72-1640 72-1662
72-1667 72-1780
72-1853
Residues/Water
72-1385 72-1417
72-1621 72-1623
72-1640 72-1643
72-1662 72-1781
Residues/Wildlife
72-1392 72-1394
72-1419 72-1438
72-1449 72-1621
72-1623 72-1640
72-1645 72-1657
72-1658 72-1659
72-1673 72-1688
72-1831 72-1839
72-1845 72-1847
Respiratory System
72-1513
Reviews
72-1598 72-1714
72-1926 72-1948
Therapeutic use
72-1481 72-1594
72-1746
Toxicity/Experimental Animals
72-1471 72-1493
72-1496 72-1512
72-1513 72-1521
72-1526 72-1527
72-1533 72-1543
72-1710 72-1732
72-1749 72-1757
72-1758 72-1772
72-1776 72-1845
72-1907 72-1908
72-1910 72-1926
72-1940 72-1964
Toxicity/Humans
72-1355 72-1380
72-1603 72-1696
72-1891 72-1926
Toxicity/Wildlife
72-1367 72-1438
72-1449 72-1598
72-1645 72-1658
72-1659 72-1669
72-1688

DDT derived compounds
Analysis
72-1781
Biochemical Effects
72-1915
Cardiovascular System
72-1911
Endocrine System
72-1911 72-1942
Factors Influencing Metabolism/Toxicity
72-1435 72-1615
72-1645 72-1845
Metabolism
72-1401 72-1487
72-1739 72-1776
72-1845 72-1911
72-1915 72-1941
Reproduction/Growth
72-1438 72-1940
Residue Degradation
72-1425 72-1776
72-1868
Residue Dynamics
72-1425
Residue/Air
72-1429
Residues/Food and Feed
72-1401 72-1402
72-1832

Residues/Humans
72-1390 72-1435
72-1615 72-1644
Residues/Water
72-1385 72-1417
72-1781
Residues/Wildlife
72-1392 72-1394
72-1419 72-1438
72-1626 72-1645
72-1845 72-1847
Toxicity/Experimental Animals
72-1776 72-1845
72-1911 72-1940
Toxicity/Wildlife
72-1438 72-1645

DDT isomers
Analysis
72-1553
Biochemical Effects
72-1471
Metabolism
72-1401 72-1489
72-1941
Nervous System
72-1471
Reproduction/Growth
72-1940
Residue Degradation
72-1425
Residue Dynamics
72-1425
Residues/Food and Feed
72-1401 72-1402
Residues/Humans
72-1644
Toxicity/Experimental Animals
72-1471 72-1940

o,p'-DDT
see DDT isomers

p,p'-DDT
see DDT

DDVP
see Dichlorvos

Demephion
see also Organophosphates
Analysis
72-2019
Residues/Food and Feed
72-2019

DFP
see also Organophosphates
Biochemical Effects
72-1929
Digestive System
72-1929
Enzyme activity
72-1477 72-1485
72-1514 72-1524
72-1766 72-1929
72-1977
Factors Influencing Metabolism/Toxicity
72-1477 72-1485
72-1514 72-1524
72-1729
Metabolism
72-1485 72-1524
Musculoskeletal System
72-1441 72-1977
Nervous System
72-1441 72-1729
Sensory System
72-1514
Toxicity/Experimental Animals
72-1441 72-1485
72-1514 72-1929
Toxicity/Humans
72-1441

Di-allate
see also Carbamates
Blood/Body Fluids
72-1922
Enzyme activity
72-1922
Factors Influencing Metabolism/Toxicity
72-1922
Integument
72-1922
Prevention
72-1452
Reproduction/Growth
72-1922
Toxicity/Experimental Animals
72-1922
Toxicity/Humans
72-1452

Dianate
see Dicamba

Diazinon
see also Organophosphates
Analysis
72-1709
Factors Influencing Metabolism/Toxicity
72-1513
Metabolism
72-1504 72-1709
Reproduction/Growth
72-1939
Residue Degradation
72-1433 72-1504
72-1849
Residues/Food and Feed
72-1433
Residues/Plants
72-1651
Residues/Soil
72-1375 72-1651
Respiratory System
72-1513
Toxicity/Experimental Animals
72-1513 72-1520

3,5-Dibromo-4-hydroxybenzonitrile
see Bromoxynil

2,6-Di-tert-butyl-4(alpha, alpha-dimethylbenzyl) phenol
Residue Degradation
72-1928
Toxicity/Experimental Animals
72-1928

2,6-Di-tert-butyl-4-nitrophenol
see also Nitro compounds
Metabolism
72-2004

Dicamba
see also Herbicides
Analysis
72-1954
Biochemical Effects
72-1954
Enzyme activity
72-1954
Metabolism
72-1865
Nervous System
72-1954
Residue Degradation
72-1865
Toxicity/Experimental Animals
72-1954

Dichlobenil
see also Herbicides
Analysis
72-1399
Metabolism
72-1864
Residue Degradation
72-1399 72-1864
Residues/Food and Feed
72-1399

Dichlofluanid
see also Fungicides
Residues/Food and Feed
72-1851

3,4-Dichloroaniline
see Propanil derived compounds

2,5-Dichloro-4-iodophenol
see Iodofenphos derived compounds

O-(2,4-Dichlorophenyl)-O,O-diethyl phosphorothioate
see Nemacide

2-(3,4-Dichlorophenyl)-4-methyl-1,2,4-

oxadiazolidine-3,5-dione
see VCS-438
5,6-Dichloro-2-trifluoromethylbenzimidazole
see also Fluorine compounds
Analysis
72-1571
Dichlorprop
see also Herbicides
Metabolism
72-1865
Residue Degradation
72-1865
Dichlorvos
see also Organophosphates
Analysis
72-1545 72-1554
72-1778
Biochemical Effects
72-1689
Cardiovascular System
72-1906
Enzyme activity
72-1527 72-1689
72-1906
Nervous System
72-1906
Prevention
72-1641
Residue Degradation
72-1554 72-1625
Residue/Air
72-1641 72-1689
Residues/Food and Feed
72-1625
Toxicity/Experimental Animals
72-1527 72-1906
Toxicity/Humans
72-1689 72-1698
Dicresyl
see also Carbamates
Prevention
72-1452
Toxicity/Humans
72-1452
Dicrotophos
see also Organophosphates
Analysis
72-1554
Metabolism
72-1753
Residue Degradation
72-1554
Dieldrin
see also Organochlorines
Analysis
72-1553 72-1781
72-1793 72-1810
72-2035
Biochemical Effects
72-1466 72-1521
72-1878
Blood/Body Fluids
72-1466
Cardiovascular System
72-1911
Cytological Effects
72-1475 72-1501
72-1902
Digestive System
72-1475 72-1486
72-1493 72-1758
72-1878 72-1902
Endocrine System
72-1466 72-1758
72-1911
Enzyme activity
72-1480 72-1484
72-1493 72-1501
72-1521 72-1878
Excretory System
72-1486 72-1902
Factors Influencing Metabolism/Toxicity
72-1475 72-1493
72-1615 72-1725
72-1845 72-1902
72-1998
Metabolism
72-1397 72-1401
72-1408 72-1431
72-1475 72-1505
72-1756 72-1845
72-1902 72-1908
72-1911 72-1913
72-1935 72-1962
72-1998
Nervous System
72-1758 72-1902
Photodecomposition
72-1842
Reproduction/Growth
72-1438 72-1486
72-1758 72-1902
72-1943
Residue Degradation
72-1383 72-1397
72-1433 72-1618
72-1842
Residue Dynamics
72-1622 72-1629
72-1756
Residues/Domestic Animals
72-1618
Residues/Food and Feed
72-1395 72-1401
72-1408 72-1428
72-1433 72-1618
72-1832
Residues/Humans
72-1380 72-1395
72-1615 72-1660
72-1878
Residues/Plants
72-1403 72-1404
72-1431
Residues/Soil
72-1383 72-1640
72-1649 72-1856
Residues/Water
72-1385 72-1622
72-1640 72-1781
Residues/Wildlife
72-1394 72-1438
72-1443 72-1626
72-1640 72-1657
72-1845
Respiratory System
72-1902
Toxicity/Experimental Animals
72-1466 72-1480
72-1486 72-1493
72-1521 72-1726
72-1754 72-1758
72-1845 72-1902
72-1908 72-1911
72-1943
Toxicity/Humans
72-1380 72-1878
Toxicity/Wildlife
72-1438 72-1443
Difolatan
see Captafol
Dimethoate
see also Organophosphates
Analysis
72-1577 72-1794
72-2006
Residue Degradation
72-1433
Residues/Domestic Animals
72-1668
Residues/Food and Feed
72-1433 72-1668
Sensory System
72-1930
Toxicity/Experimental Animals
72-1930
Toxicity/Humans
72-1930
m-(N,N-Dimethylformamidine) phenyl N-methylcarbamate
see also Carbamates
Photodecomposition
72-1424
O,O-Dimethyl-S-isopropyl thioethyl phosphorodithioate
see Z-7272
3,4-Dimethylphenyl N-methylcarbamate

Analysis
72-1549
1,1-Dimethyl-3-(alpha, alpha,alpha-trifluoro-m-tolyl) urea
see Fluometuron
Dioxathion
see also Organophosphates
Enzyme activity
72-1519
Factors Influencing Metabolism/Toxicity
72-1519
Toxicity/Experimental Animals
72-1519
Dioxins
see also 2,4,5-T
Analysis
72-1416 72-1572
Blood/Body Fluids
72-1535
Cardiovascular System
72-1536
Digestive System
72-1536
Endocrine System
72-1536
Environmental Pollution
72-1355 72-1608
Enzyme activity
72-1535
Musculoskeletal System
72-2003
Mutagenesis/ Teratogenesis
72-1745 72-2003
Residues/Wildlife
72-1416
Respiratory System
72-1536
Toxicity/Experimental Animals
72-1535 72-1536 72-2003
Toxicity/Humans
72-1355
Diphenamid
see also Herbicides
Analysis
72-2026
Residues/Food and Feed
72-1413
Residues/Plants
72-1413
Diptal
see Di-allate
Diquat
see also Herbicides
Analysis
72-1547
Digestive System
72-1770
Education/Training
72-1354
Enzyme activity
72-1770
Excretory System
72-1770
Laws and Regulations
72-1354
Metabolism
72-1717 72-2000
Photodecomposition
72-1717
Prevention
72-1354
Residues/Soil
72-1547
Respiratory System
72-1770 72-2000
Toxicity/Experimental Animals
72-1770 72-2000
Disulfiram
see also Fungicides
Cardiovascular System
72-1462
Factors Influencing Metabolism/Toxicity
72-1462
Toxicity/Experimental Animals
72-1462
Disulfoton
see also Organophosphates
Residues/Food and Feed
72-1653
Diuron
see also Herbicides
Analysis
72-1800
DNOC
see also Nitro compounds
Residues/Food and Feed
72-1665
Dursban
see also Organophosphates
Enzyme activity
72-1728
Factors Influencing Metabolism/Toxicity
72-1728
Toxicity/Experimental Animals
72-1728
Dyfonate
see also Organophosphates
Enzyme activity
72-1947
Metabolism
72-1947
Residues/Plants
72-1651
Residues/Soil
72-1651 72-1867
Endosulfan
see also Organochlorines
Factors Influencing Metabolism/Toxicity
72-1513
Metabolism
72-1530 72-1913
Photodecomposition
72-1842
Residue Degradation
72-1393 72-1842
Residue Removal
72-1393
Residues/Water
72-1393
Respiratory System
72-1513
Toxicity/Experimental Animals
72-1513
Endrin
see also Organochlorines
Analysis
72-1553 72-1555 72-1781 72-1783 72-1793 72-2013
Beneficial Effects
72-1367
Biochemical Effects
72-1521 72-1878
Digestive System
72-1732 72-1878
Environmental Pollution
72-1367
Enzyme activity
72-1521 72-1878
Excretory System
72-1683 72-1732
Metabolism
72-1401 72-1405 72-1908 72-1913 72-1962
Musculoskeletal System
72-1683
Nervous System
72-1439
Photodecomposition
72-1555
Reproduction/Growth
72-1319
Residue Degradation
72-1433
Residues/Food and Feed
72-1401 72-1405 72-1433
Residues/Humans
72-1660 72-1878
Residues/Plants
72-1403 72-2013
Residues/Water
72-1781

Toxicity/Experimental
Animals
72-1521 72-1732
72-1908 72-1919
Toxicity/Humans
72-1439 72-1683
72-1878
Toxicity/Wildlife
72-1367
Endrin derived compounds
Analysis
72-1555 72-1783
Photodecomposition
72-1555
EPN
see also
Organophosphates
Analysis
72-1552
Enzyme activity
72-1485
Factors Influencing
Metabolism/Toxicity
72-1485
Metabolism
72-1485
Toxicity/Experimental
Animals
72-1485
Toxicity/Humans
72-1450
Treatment of Poisoning
72-1450
Eptam
see EPTC
EPTC
see also Carbamates
Prevention
72-1452
Toxicity/Humans
72-1452
Erbon
see also Herbicides
Biochemical Effects
72-1966
Endocrine System
72-1966
Prevention
72-1966
Toxicity/Experimental
Animals
72-1966
Ethephon
see also Herbicide,
Herbicides
Factors Influencing
Metabolism/Toxicity
72-1903 72-1914
Metabolism
72-1472 72-1903
72-1914
Reproduction/Growth
72-1472
Ethion
see also
Organophosphates
Analysis
72-2006
Residue Degradation
72-1433
Residues/Food and Feed
72-1433
Toxicity/Experimental
Animals
72-1520
Ethylenethiourea
see ETU
S-Ethyl hexamethylene carbamate
see Molinate
Ethyl mercury chloride
see Granosan
Ethylmethylfluorothiophosp
Enzyme activity
72-1528
Metabolism
72-1528
Toxicity/Experimental
Animals
72-1528
Ethyl parathion
see Parathion
O-Ethyl S-phenyl ethylphosphonodithioate
see Dyfonate
ETU
Endocrine System
72-2005
Fenazaflor
see also Fluorine
compounds
Analysis
72-1571 72-2032
72-2033
Residues/Food and Feed
72-2032 72-2033
Fenitrothion
see also
Organophosphates
Analysis
72-1789
Enzyme activity
72-1527 72-1730
Factors Influencing
Metabolism/Toxicity
72-1734
Metabolism
72-1516
Nervous System
72-1985
Residue Degradation
72-1849
Residues/Food and Feed
72-1409 72-1516
72-1537 72-1789
Sensory System
72-1730
Toxicity/Experimental
Animals
72-1516 72-1527
72-1537 72-1730
72-1733 72-1734
72-1985
Fenoprop
see also Herbicides
Metabolism
72-1865
Residue Degradation
72-1865
Fensulfothion
see also
Organophosphates
Residues/Food and Feed
72-1426
Residues/Plants
72-1426
Fenthion
see also Avicides,
Organophosphates
Enzyme activity
72-1730
Sensory System
72-1730
Toxicity/Experimental
Animals
72-1730 72-1937
Fluenethyl
see also Fluorine
compounds
Analysis
72-2031
Residues/Food and Feed
72-2031
Fluometuron
see also Herbicides
Analysis
72-2025
Biochemical Effects
72-1966
Endocrine System
72-1966
Prevention
72-1966
Residue Degradation
72-1415
Toxicity/Experimental
Animals
72-1966
Fluorine compounds
see also 5,6-Dichloro-2-trifluoromethylbenzimidazole, Fenazaflor, Fluenethyl, Fluoroacetamide, MNFA
Fluoroacetamide
see also Fluorine
compounds, Rodenticides
Analysis
72-1967
Biochemical Effects
72-1968

Blood/Body Fluids
72-1968
Digestive System
72-1968
Metabolism
72-1967
Toxicity/Experimental Animals
72-1967 72-1968

Fluorodifen
see also Herbicides
Metabolism
72-1983

Formothion
see also Organophosphates
Metabolism
72-1503
Residues/Food and Feed
72-1434 72-1503

Fosfotion
see Malathion

Fumigants
see also Aluminum phosphide, Chloropicrin, Metham, Methyl bromide, Phosphine

Fundal
see Chlordimeform

Fungicides
see also Agallol, Benomyl, Biphenyl, Captafol, Captan, Copper sulfate, Dichlofluanid, Disulfiram, Hexachlorobenzene, Hinosan, Maneb, Mercurials, Pentachlorobenzaldoxime, Polyoxin D, Sodium diethyldithiocarbamate, Thiophanate, Tri-o-cresyl phosphate
Analysis
72-1566
Metabolism
72-1948
Reviews
72-1948
Toxicity/Wildlife
72-1892

Fussol
see Fluoroacetamide

Galecron
see Chlordimeform

Gardona
see Tetrachlorvinphos

GA-3
see also Organophosphates
Biochemical Effects
72-1760
Nervous System
72-1760
Toxicity/Experimental Animals
72-1760

Gramoxone
see Paraquat

Granosan
see also Mercurials
Metabolism
72-1769

Granosan M
see Mercurials

Halowax
see Chlorinated naphthalenes

Hempa
see also Chemosterilants
Factors Influencing Metabolism/Toxicity
72-1743
Toxicity/Experimental Animals
72-1743

Heptachlor
see also Organochlorines
Analysis
72-1781 72-1810
Biochemical Effects
72-1521
Cytological Effects
72-1539 72-1737
Digestive System
72-1464 72-1539
72-1737
Endocrine System
72-1737
Enzyme activity
72-1464 72-1521
Excretory System
72-1737
Factors Influencing Metabolism/Toxicity
72-1464 72-1737
Metabolism
72-1401 72-1405
72-1431 72-1716
Mutagenesis/Teratogenesis
72-1464
Photodecomposition
72-1871
Reproduction/Growth
72-1464
Residue Degradation
72-1433 72-1618
Residues/Domestic Animals
72-1432 72-1618
Residues/Food and Feed
72-1401 72-1405
72-1432 72-1433
72-1618
Residues/Humans
72-1660
Residues/Plants
72-1431
Residues/Water
72-1781
Toxicity/Experimental Animals
72-1464 72-1521
72-1737

Heptachlor epoxide
see also Organochlorines
Analysis
72-1781 72-1810
Digestive System
72-1934
Enzyme activity
72-1934
Metabolism
72-1401
Residue Degradation
72-1618
Residues/Domestic Animals
72-1618
Residues/Food and Feed
72-1401 72-1618
Residues/Humans
72-1378
Residues/Soil
72-1650
Residues/Water
72-1781

Herbicides
see also Alachlor, Ametryne, Amitrole, Ammonium Thiocyanate, Amo 1618, Arsenicals, Atrazine, Bandane, Barban, Benefin
see also Bensulide, Bromacil, Bromoxynil, Chlorbromuron, Chlormequat chloride, Chlorpropham, Chlorthiamid, Dalapon, Dicamba, Dichlobenil, Dichlorprop, Diphenamid, Diquat, Diuron, Erbon, Ethephon, Fenoprop, Fluometuron, Fluorodifen, Maleic hydrazide, MCCP, MCPA, MCPB, Methomyl, Metobromuron, Molinate, Monuron, Nitralin, Paraquat, PCP, Phosfon, Picloram, Prometryne, Propachlor, Propham, Simazine, Sodium chlorate, 2,3,6-TBA, TCA, TCA, VCS-438
Analysis
72-1546 72-1547
72-1566 72-1572
72-1632 72-2015

72-2018
Environmental Pollution
72-1366
Enzyme activity
72-1502
Metabolism
72-1923
Mutagenesis/
Teratogenesis
72-1509
Residue Degradation
72-1502 72-1633
Residue Dynamics
72-1374
Residues/Soil
72-1547 72-1632
72-1633 72-2018
Residues/Water
72-1859
Reviews
72-1859
Toxicity/Wildlife
72-1892
Hexachlorane
see BHC
Hexachlorobenzene
see also Fungicides
Residues/Food and Feed
72-1395
Residues/Humans
72-1395
1,4,5,6,7,7-Hexachloro-norbornene-2,3-bis-chloromethylene
Metabolism
72-1530
Hexamethylphosphoramide
see Hempa
Hinosan
see also Fungicides
Prevention
72-1701
Toxicity/Humans
72-1701
Hydram
see Molinate
2-Hydroxyatrazine
see Atrazine
Hydroxydiazinon
see Diazinon
Hydroxyisoxazole
Residues/Food and Feed
72-1411
3-Hydroxy-N-methoxy-N-methyl-cis-crotonamide dimethyl phosphate
see C-2307
3-hydroxy-5-methyl isoxazole
see Hydroxyisoxazole
IBP
see Kitazin-P
Imidan
see Phosmet
Iodofenoxon
see Iodofenphos derived compounds
Iodofenphos
see also
Organophosphates
Analysis
72-2037
Residues/Food and Feed
72-2037
Iodofenphos derived compounds
Analysis
72-2037
Residues/Food and Feed
72-2037
Isobornyl thiocyanoacetate
see Thanite
Isodrin
see also Organochlorines
Analysis
72-1793
Isopropyl carbanilate
see Propham
Isopropylmethylfluorcthiop
Enzyme activity
72-1528
Metabolism
72-1528
Toxicity/Experimental Animals
72-1528
Isopropyl-methylphosphonofluoridate
see Sarin
Kepone
see Chlordeeone
Kikuthrin
see Proparthrin
Kitazin-P
see also
Organophosphates
Residues/Food and Feed
72-1381
Landrin
see also Carbamates
Analysis
72-1573
Lannate
see Methomyl
Lead
Metabolism
72-1400
Residues/Food and Feed
72-1400
Reviews
72-1680
Treatment of Poisoning
72-1680
Lindane
see also Organochlorines
Analysis
72-1575 72-1781
72-1791 72-1810
Biochemical Effects
72-1521
Digestive System
72-1732
Enzyme activity
72-1521 72-1774
Excretory System
72-1732
Factors Influencing Metabolism/Toxicity
72-1513 72-1988
Metabolism
72-1490 72-1935
72-1988 72-1989
Prevention
72-1888
Reproduction/Growth
72-1919 72-1939
Residue Degradation
72-1791
Residue Dynamics
72-1622
Residue Removal
72-1841
Residues/Food and Feed
72-1382 72-1395
72-1666 72-1667
Residues/Humans
72-1378 72-1390
72-1395
Residues/Soil
72-1667
Residues/Water
72-1385 72-1622
72-1781
Residues/Wildlife
72-1847
Respiratory System
72-1513
Reviews
72-1888
Toxicity/Experimental Animals
72-1513 72-1521
72-1732 72-1888
72-1919
Toxicity/Humans
72-1888
Treatment of Poisoning
72-1888
Linuron
see also Herbicides
Biochemical Effects
72-1966
Endocrine System
72-1966
Prevention
72-1966
Toxicity/Experimental Animals
72-1966
Lovozal
see Fenazaflor

Malaoxon
see also
Organophosphates
Enzyme activity
72-1511
Metabolism
72-1724
Malathion
see also
Organophosphates
Analysis
72-1544 72-1778
72-1779 72-2006
Biochemical Effects
72-1532 72-1731
Digestive System
72-1532
Education/Training
72-1354
Enzyme activity
72-1445 72-1532
Factors Influencing
Metabolism/Toxicity
72-1513
Laws and Regulations
72-1354
Metabolism
72-1469 72-1724
72-1935
Mutagenesis/
Teratogenesis
72-1971
Nervous System
72-1731
Prevention
72-1354 72-1445
72-1887
Reproduction/Growth
72-1939
Residue Degradation
72-1387 72-1469
72-1625 72-1652
72-1849
Residue Removal
72-1887
Residues/Food and Feed
72-1387 72-1427
72-1625 72-1652
72-1851
Respiratory System
72-1513
Reviews
72-1971
Sensory System
72-1445
Toxicity/Experimental
Animals
72-1513 72-1532
Toxicity/Humans
72-1445

Maleic hydrazide
see also Herbicides
Mutagenesis/
Teratogenesis
72-1890
Maneb
see also Fungicides
Analysis
72-2009
Endocrine System
72-2005
Prevention
72-1452
Toxicity/Humans
72-1452
MCPA
see also Herbicides
Metabolism
72-1865 72-2001
Residue Degradation
72-1865
MCPB
see also Herbicides
Metabolism
72-1865 72-2001
Residue Degradation
72-1865
MCPP
see also Herbicides
Metabolism
72-1865
Residue Degradation
72-1865
Mecoprop
see MCPP
Melipax
see Toxaphene
MEMC
Enzyme activity
72-1459
Excretory System
72-1459
Toxicity/Experimental
Animals
72-1459
Menazon
see also
Organophosphates
Analysis
72-1582
Prevention
72-1448
Residues/Humans
72-1582
MEP
see Fenitrothion
Mercurials
see also Fungicides,
Agallol, Granosan, PMA
Analysis
72-1786 72-1803
72-2015 72-2020
72-2021
Digestive System
72-1440 72-1464
Environmental Pollution
72-1605 72-1609
72-1828
Enzyme activity
72-1464 72-1693
Factors Influencing
Metabolism/Toxicity
72-1464
Integument
72-1449
Laws and Regulations
72-1359
Metabolism
72-1712 72-1846
Morbidity and Mortality
Statistics
72-1693
Mutagenesis/
Teratogenesis
72-1449 72-1464
Nervous System
72-1440 72-1880
Reproduction/Growth
72-1438 72-1449
72-1464 72-1631
72-1669 72-1692
72-1712
Residues/Food and Feed
72-1846
Residues/Humans
72-1631 72-1838
Residues/Wildlife
72-1438 72-1449
72-1831 72-1844
Reviews
72-1605 72-1609
72-1680 72-1828
Sensory System
72-1693
Toxicity/Experimental
Animals
72-1464 72-1712
Toxicity/Humans
72-1440 72-1692
72-1693 72-1880
72-1882 72-1886
Toxicity/Wildlife
72-1438 72-1449
72-1605 72-1669
Treatment of Poisoning
72-1680 72-1886
Mesomil
see Methomyl
Mesurol
see Methiocarb
Metallic compounds
Analysis
72-1578
Reviews
72-1578
Metham
see also Fumigants
Reproduction/Growth
72-1761
Residue Degradation
72-1761

Methidathion
see also
Organophosphates
Toxicity/Experimental
Animals
72-1520
Methiocarb
see also Carbamates
Analysis
72-1573
Methomyl
see also Carbamates,
Herbicides
Analysis
72-2036
Residues/Food and Feed
72-1410
Methoxychlor
see also Organochlorines
Factors Influencing
Metabolism/Toxicity
72-1708
Metabolism
72-1708
Toxicity/Experimental
Animals
72-1708
Methoxyethyl mercuric chloride
see MEMC
Methoxyethylmercury chloride
see Agallol
Methyl bromide
see also Fumigants
Analysis
72-2010 72-2034
Residues/Food and Feed
72-1396
Toxicity/Experimental
Animals
72-1747
Treatment of Poisoning
72-1747
O-Methyl-O-cyclohexyl S-(4-chlorophenyl) thiophosphate
see MHCP
Methyl mercaptophos
see also
Organophosphates
Analysis
72-1805
Blood/Body Fluids
72-1542
Immunology
72-1771
Respiratory System
72-1542
Sensory System
72-1542
Toxicity/Experimental
Animals
72-1542
Methylnitrophos
see Fenitrothion
Methyl parathion
see also
Organophosphates
Analysis
72-1789
Enzyme activity
72-1999
Metabolism
72-1999
Reproduction/Growth
72-1761
Residue Degradation
72-1433 72-1761
Residues/Food and Feed
72-1433 72-1789
Metobromuron
see also Herbicides
Analysis
72-2025
Metribuzin
see BAY 94337
Mevinphos
see also
Organophosphates
Analysis
72-1554
Cardiovascular System
72-1906
Enzyme activity
72-1906
Nervous System
72-1906
Residue Degradation
72-1554
Toxicity/Experimental
Animals
72-1906
MHCP
see also
Organophosphates
Toxicity/Humans
72-1450
Treatment of Poisoning
72-1450
Minokol
see
Pentachlorobenzaldoxime
Mipafox
Enzyme activity
72-1718
Mirex
see also Organochlorines
Analysis
72-1560
Environmental Pollution
72-1355
Toxicity/Humans
72-1355
MNPA
see also Fluorine compounds
Analysis
72-1967
Biochemical Effects
72-1968
Blood/Body Fluids
72-1968
Cardiovascular System
72-1969
Digestive System
72-1968
Metabolism
72-1967
Nervous System
72-1899
Toxicity/Experimental
Animals
72-1967 72-1968
72-1969
Toxicity/Humans
72-1969
Moban
see also Carbamates
Metabolism
72-1707
Molinate
see also Carbamates,
Herbicides
Prevention
72-1452
Toxicity/Humans
72-1452
Monocrotophos
Analysis
72-1554
Residue Degradation
72-1554
Monuron
see also Herbicides
Factors Influencing
Metabolism/Toxicity
72-1513
Respiratory System
72-1513
Toxicity/Experimental
Animals
72-1513
MPMC
see 3,4-Dimethylphenyl N-methylcarbamate
MC-2983
see 5,6-Dichloro-2-trifluoromethylbenzimida
Nemacide
see also
Organophosphates
Analysis
72-1508
Enzyme activity
72-1508
Metabolism
72-1508
Toxicity/Experimental
Animals
72-1508

NH-6967
see also Carbamates
Residues/Food and Feed
72-1411
Nippon
Prevention
72-1448
Nissol
see MNPA
Nitralin
see also Herbicides
Residues/Food and Feed
72-1411
Nitro compounds
see also 2,6-Di-tert-butyl-4-nitrophenol, DNOC, PCNB
4-Nitro-m-cresol
see Fenitrothion
Organochlorines
see also Aldrin, BHC, Chlorbicyclen, Chlordane, Chlordane, DDT, Dieldrin, Dieldrin, Endrin, Heptachlor, Heptachlor epoxide, Isodrin, Lindane, Methoxychlor, Mirex, Polychlorinated biphenyls, TDE, Toxaphene, alpha-Trichloromethyl-p-ethoxybenzyl-p-ethoxyaniline
Analysis
72-1416 72-1556
72-1557 72-1558
72-1559 72-1561
72-1567 72-1574
72-1581 72-1639
72-1777 72-1785
72-1787 72-1790
72-1799 72-1804
72-1806 72-2014
72-2015 72-2020
72-2023 72-2024
Carcinogenesis
72-1926
Environmental Pollution
72-1588 72-1601
72-1605 72-1613
72-1822 72-1828
Enzyme activity
72-1693
Factors Influencing Metabolism/Toxicity
72-1454 72-1674
Integument
72-1451
Laws and Regulations
72-1600
Metabolism
72-1958 72-1965
Morbidity and Mortality Statistics
72-1454 72-1681
72-1693
Nervous System
72-1442
Photodecomposition
72-1671 72-1777
Reproduction/Growth
72-1893
Residue Degradation
72-1671
Residues/Domestic Animals
72-1895
Residues/Food and Feed
72-1379 72-1557
72-1588 72-1619
72-1646 72-1837
72-1852
Residues/Humans
72-1639 72-1664
72-1674 72-1875
Residues/Soil
72-1670
Residues/Water
72-1613 72-1806
Residues/Wildlife
72-1416
Reviews
72-1442 72-1605
72-1613 72-1646
72-1828 72-1895
72-1926 72-1958
Sensory System
72-1451 72-1693
Toxicity/Domestic Animals
72-1883 72-1894
72-1895
Toxicity/Experimental Animals
72-1926
Toxicity/Humans
72-1442 72-1451
72-1454 72-1681
72-1693 72-1700
72-1705 72-1926
Toxicity/Wildlife
72-1605 72-1893
Treatment of Poisoning
72-1451 72-1883
Organophosphates
see also Amiton, Azinphosmethyl, Coumaphos, Crufomate, C-2307, Demephion, DFP, Diazinon, Dichlorvos, Dicrotophos, Dimethoate, Dioxathion, Disulfoton, Dursban, Dyfonate, EPN, Ethion, Fenitrothion, Fensulfothion, Fenthion, Formothion, GA-3, Iodofenphos, Kitazin-P, Malaoxon, Malathion, Menazon, Methidathion, Methyl mercaptophos, Methyl parathion, Mevinphos, MHCP, Nemacide, Parathion, Phorate, Phosmet, Ronnel, Ronnel, Sarin, Schradan, Soman, S 4087, TEPP, Tetrachlorvinphos, Trichlorfon, Tri-o-cresyl phosphate, Triethylphosphate, Vamidothion
Analysis
72-1556 72-1566
72-1578 72-1579
72-1790 72-1798
72-2015 72-2020
Biochemical Effects
72-1682 72-1689
Blood/Body Fluids
72-1721
Cardiovascular System
72-1898
Digestive System
72-1898
Environmental Pollution
72-1361 72-1605
72-1843
Enzyme activity
72-1531 72-1682
72-1689 72-1693
72-1706 72-1721
72-1765 72-1920
72-1957
Metabolism
72-1461 72-1840
72-1920 72-1948
72-1958 72-1961
Morbidity and Mortality Statistics
72-1444 72-1681
72-1693 72-1900
Nervous System
72-1442 72-1525
72-1703 72-1765
72-1898 72-1920
72-1957
Prevention
72-1879
Residue Degradation
72-1843 72-1961
Residue/Air
72-1689
Residues/Food and Feed
72-1840 72-1852
72-1961
Reviews
72-1442 72-1461
72-1578 72-1605
72-1798 72-1840
72-1843 72-1896

72-1948 72-1958
72-1961
Sensory System
72-1444 72-1453
72-1531 72-1691
72-1693 72-1695
72-1703 72-1898
Toxicity/Domestic Animals
72-1695 72-1883
72-1894 72-1896
Toxicity/Experimental Animals
72-1461 72-1518
72-1525 72-1531
72-1920
Toxicity/Humans
72-1442 72-1444
72-1453 72-1461
72-1531 72-1676
72-1681 72-1682
72-1689 72-1691
72-1693 72-1703
72-1898 72-1900
Toxicity/Wildlife
72-1605
Treatment of Poisoning
72-1444 72-1453
72-1706 72-1721
72-1883 72-1896

Paraoxon
Enzyme activity
72-1485 72-1977
Factors Influencing Metabolism/Toxicity
72-1485
Metabolism
72-1485
Musculoskeletal System
72-1977
Toxicity/Experimental Animals
72-1485

Paraquat
see also Herbicides
Analysis
72-1547
Biochemical Effects
72-1467
Digestive System
72-1446 72-1770
72-1992
Education/Training
72-1354
Enzyme activity
72-1770
Excretory System
72-1770
Laws and Regulations
72-1354
Metabolism
72-2000
Prevention
72-1354 72-1448
Reproduction/Growth
72-1467
Residues/Soil
72-1547
Respiratory System
72-1446 72-1770
72-1897 72-1917
72-1918 72-2000
Toxicity/Experimental Animals
72-1770 72-1917
72-1918 72-1992
72-2000
Toxicity/Humans
72-1446 72-1694
72-1897
Treatment of Poisoning
72-1446

Parathion
see also Organophosphates
Analysis
72-1384 72-1554
72-1795
Biochemical Effects
72-1437 72-1731
Blood/Body Fluids
72-1964
Cytological Effects
72-1924
Digestive System
72-1437 72-1468
72-1909
Environmental Pollution
72-1603
Enzyme activity
72-1437 72-1468
72-1484 72-1485
72-1677 72-1699
72-1903 72-1951
72-1964 72-1999
Factors Influencing Metabolism/Toxicity
72-1437 72-1454
72-1485 72-1496
72-1964
Metabolism
72-1468 72-1485
72-1795 72-1909
72-1935 72-1951
72-1964 72-1999
Morbidity and Mortality Statistics
72-1454 72-1881
Musculoskeletal System
72-1437
Nervous System
72-1437 72-1731
Prevention
72-1887
Reproduction/Growth
72-1943 72-1964
Residue Degradation
72-1377 72-1433
72-1554 72-1648
Residue Removal
72-1887
Residues/Food and Feed
72-1384 72-1433
Residues/Humans
72-1675
Respiratory System
72-1437
Sensory System
72-1437
Toxicity/Experimental Animals
72-1485 72-1496
72-1903 72-1924
72-1943 72-1964
Toxicity/Humans
72-1437 72-1450
72-1454 72-1603
72-1675 72-1699
72-1700
Treatment of Poisoning
72-1437 72-1450
72-1699

PCNB
see also Nitro compounds
Analysis
72-1786

PCP
see also Herbicides
Analysis
72-1575
Biochemical Effects
72-1463
Metabolism
72-1495 72-1959
Nervous System
72-1439
Residue Degradation
72-1959
Toxicity/Experimental Animals
72-1463
Toxicity/Humans
72-1439

Pentachlorobenzaldoxime
see also Fungicides
Analysis
72-1801

Pentachlorobiphenyl
see Polychlorinated biphenyls

Phenylmercuric acetate
see PMA

Phorate
see also Organophosphates
Analysis
72-2006
Residue Degradation
72-1849

Phosfon
see also Herbicides

Biochemical Effects
72-1764
Toxicity/Experimental Animals
72-1764
Phosmet
see also Organophosphates
Enzyme activity
72-1519
Factors Influencing Metabolism/Toxicity
72-1519
Toxicity/Experimental Animals
72-1519
Phosphamide
see Dimethoate
Phosphine
see also Fumigants
Residue Removal
72-1634
Residues/Food and Feed
72-1634
Phostoxin
see Aluminum phosphide
Photoendrin
see Endrin derived compounds
Picloram
see also Herbicides
Analysis
72-2027
Digestive System
72-1950
Education/Training
72-1354
Environmental Pollution
72-1823
Laws and Regulations
72-1354
Nervous System
72-1950
Prevention
72-1354
Reproduction/Growth
72-2002
Residues/Soil
72-1857
Toxicity/Experimental Animals
72-1950
Pielik
see 2,4-D
Piperonyl butoxide
see also Synergists
Blood/Body Fluids
72-1933
Cytological Effects
72-1470 72-1994
Enzyme activity
72-1994
Factors Influencing Metabolism/Toxicity
72-1736 72-1938
Metabolism
72-1742
Toxicity/Experimental Animals
72-1470 72-1736
72-1933 72-1938
Toxicity/Humans
72-1736
Planavin
see Nitralin
Plictran
see also Tin compounds
Residues/Food and Feed
72-1874
PMA
see also Mercurials
Mutagenesis/Teratogenesis
72-1971
Reviews
72-1971
Polychlorinated biphenyls
see also Organochlorines
Analysis
72-1416 72-1563
72-1639 72-1782
72-1785 72-1796
72-2008 72-2011
72-2017 72-2028
72-2029
Environmental Pollution
72-1355
Enzyme activity
72-1488 72-1978
Factors Influencing Metabolism/Toxicity
72-1615 72-1708
Integument
72-1449
Laws and Regulations
72-1607
Metabolism
72-1616 72-1708
72-1776 72-1782
Musculoskeletal System
72-1978
Mutagenesis/Teratogenesis
72-1449
Reproduction/Growth
72-1449 72-1483
72-1491 72-1598
72-1669
Residue Degradation
72-1776
Residue/Air
72-1429
Residues/Food and Feed
72-1420
Residues/Humans
72-1430 72-1615
72-1639
Residues/Wildlife
72-1416 72-1419
72-1449 72-1616
72-1657 72-1831
72-2017
Reviews
72-1598 72-2017
Toxicity/Experimental Animals
72-1488 72-1491
72-1708 72-1776
72-1978
Toxicity/Humans
72-1355 72-1700
Toxicity/Wildlife
72-1449 72-1598
72-1669
Polychlorinated terphenyls
Residues/Wildlife
72-1418
Polychlorobenzofurans
Analysis
72-1416
Residues/Wildlife
72-1416
Polyoxin D
see also Fungicides
Analysis
72-1551
Pralidoxime
see also Antidotes
Metabolism
72-1925
Preforan
see Fluorodifen
Prometryne
see also Herbicides
Analysis
72-2006
Propachlor
see also Herbicides
Residues/Soil
72-1375
Propanil derived compounds
Metabolism
72-1984
Residue Degradation
72-1423
Proparthrin
see also Pyrethrins
Photodecomposition
72-1834
Residue Degradation
72-1834
Propham
see also Herbicides
Metabolism
72-1872
Residues/Domestic Animals
72-1872
Propoxur
see also Carbamates
Analysis
72-1573

S-Propyl dipropylthiocarbamate
see Vanalate
Pyrethrins
see also Botanicals, Proparthrin
Blood/Body Fluids 72-1933
Digestive System 72-1498
Enzyme activity 72-1498
Factors Influencing Metabolism/Toxicity 72-1628
Immunology 72-1936
Integument 72-1936
Metabolism 72-1735
Toxicity/Experimental Animals 72-1933 72-1936
Pyrethrum
see Pyrethrins
Pyrilidinyl-2,4-dichlorophenyl carbamate
see NH-6967
Queletox
see Fenthion
Rabon
see Tetrachlorvinphos
Reglon
see Diquat
Reglone
see Diquat
Rodenticides
see also Fluoroacetamide, Fluoroacetamide, Thallium, Warfarin, Zinc phosphide
Morbidity and Mortality Statistics 72-1455
Prevention 72-1455
Toxicity/Domestic Animals 72-1455
Rogor
see Dimethoate
Ronnel
see also Organophosphates
Biochemical Effects 72-1762
Blood/Body Fluids 72-1762
Therapeutic use 72-1762
Rotenone
see also Botanicals
Analysis 72-1808
Enzyme activity 72-1719
Factors Influencing Metabolism/Toxicity 72-1719
Photodecomposition 72-1870
Toxicity/Experimental Animals 72-1482 72-1719
Ruelene
see Crufomate
R-3828
see also Organophosphates
Metabolism 72-1517
Sarin
see also Organophosphates
Blood/Body Fluids 72-1975
Enzyme activity 72-1974 72-1975 72-1996
Factors Influencing Metabolism/Toxicity 72-1975
Nervous System 72-1974 72-1996
Sayfos
see Menazon
Schradan
see also Organophosphates
Analysis 72-1778
Sesamex
see also Synergists
Enzyme activity 72-1484
Sevin
see Carbaryl
Simazine
see also Herbicides
Metabolism 72-1956
Prevention 72-1448
Reproduction/Growth 72-1927
Residue Degradation 72-1861
Residues/Food and Feed 72-1861
Residues/Soil 72-1861
Sodium arsenate
Mutagenesis/ Teratogenesis 72-1971
Reviews 72-1971
Sodium arsenite
see also Arsenicals
Mutagenesis/ Teratogenesis 72-1494
Reproduction/Growth 72-1494
Residues/Food and Feed 72-1656
Sodium chlorate
see also Herbicides
Reviews 72-1680
Treatment of Poisoning 72-1680
Sodium diethyldithiocarbamate
see also Fungicides
Cardiovascular System 72-1462
Factors Influencing Metabolism/Toxicity 72-1462
Toxicity/Experimental Animals 72-1462
Sodium ethyl xanthogenate
Reproduction/Growth 72-1761
Residue Degradation 72-1761
Soman
see also Organophosphates
Biochemical Effects 72-1929
Digestive System 72-1929
Enzyme activity 72-1929 72-1953
Excretory System 72-1953
Musculoskeletal System 72-1953
Nervous System 72-1953
Toxicity/Experimental Animals 72-1929
Treatment of Poisoning 72-1953
Strychnine
see also Rodenticides
Nervous System 72-1740
Toxicity/Experimental Animals 72-1740
Sumithion
see Fenitrothion
Supracide
see Methidathion
Synergists
see also Piperonyl

butoxide, Sesamex
S 4087
see also
Organophosphates
Enzyme activity
72-1944
Reproduction/Growth
72-1944
Toxicity/Experimental
Animals
72-1944

2,4,5-T
see also Herbicides
Education/Training
72-1354
Environmental Pollution
72-1608 72-1829
Laws and Regulations
72-1354
Metabolism
72-2001
Musculoskeletal System
72-1995 72-2003
Mutagenesis/
Teratogenesis
72-1678 72-1745
72-1995 72-2003
Prevention
72-1354
Reproduction/Growth
72-1991
Reviews
72-1678
Toxicity/Experimental
Animals
72-1991 72-1995
72-2003

2,4,5-TB
Metabolism
72-2001

2,3,6-TBA
see also Herbicides
Analysis
72-1954
Biochemical Effects
72-1954
Enzyme activity
72-1954
Metabolism
72-1617
Nervous System
72-1954
Residue Degradation
72-1617
Toxicity/Experimental
Animals
72-1954

TCA
see also Herbicides
Reproduction/Growth
72-1927

TCDD
see Dioxins
TDE
see also Organochlorines
Analysis
72-1781 72-2013
Biochemical Effects
72-1538 72-1722
72-1767
Carcinogenesis
72-1481
Endocrine System
72-1538 72-1976
Enzyme activity
72-1481 72-1538
72-1767 72-1976
Factors Influencing
Metabolism/Toxicity
72-1615 72-1645
Nervous System
72-1722
Reproduction/Growth
72-1438
Residue Degradation
72-1868
Residue/Plants
72-2013
Residues/Humans
72-1615
Residues/Water
72-1385 72-1781
Residues/Wildlife
72-1392 72-1394
72-1438 72-1645
Reviews
72-1460
Therapeutic use
72-1460 72-1479
72-1481 72-1529
72-1540 72-1763
72-1904 72-1905
72-1970
Toxicity/Experimental
Animals
72-1538 72-1722
Toxicity/Wildlife
72-1438 72-1645

Telodrin
Biochemical Effects
72-1521
Enzyme activity
72-1521
Toxicity/Experimental
Animals
72-1521

TEPP
see also
Organophosphates
Enzyme activity
72-1477 72-1485
Factors Influencing
Metabolism/Toxicity
72-1477 72-1485
Metabolism
72-1485
Toxicity/Experimental
Animals
72-1485
Terrachlor
see PCNB
2,3,7,8-tetrachlorobenzo-
p-dioxin
see Dioxins
2,3,7,8-
tetrachlorodibenzo-p-
dioxin
see Dioxins
2,3,7,8-tetrachloro-p-
dioxin
see Dioxins
N-
Tetrachloroethylthiotetrah
Nervous System
72-1439
Toxicity/Humans
72-1439
Tetrachlorvinphos
see also
Organophosphates
Residues/Domestic
Animals
72-1727
Residues/Food and Feed
72-1727
Toxicity/Experimental
Animals
72-1727
Tetraethylpyrophosphate
see TEPP
Tetram
see Amiton

Thallium
see also Rodenticides
Nervous System
72-1436 72-1697
72-1884
Toxicity/Humans
72-1436 72-1697
72-1884
Treatment of Poisoning
72-1436

Thanite
Biochemical Effects
72-1945
Blood/Body Fluids
72-1945
Toxicity/Experimental
Animals
72-1945

Thiodan
see Endosulfan

Thiophanate
see also Fungicides
Metabolism
72-1921

Thio-TEPA
Mutagenesis/
Teratogenesis
72-1971
Reviews
72-1971
Tin compounds
see also Plictran
TOCP
see Tri-o-cresyl
phosphate
Tordon
see Picloram
TOTP
see Tri-o-cresyl
phosphate
Toxaphene
see also Organochlorines
Analysis
72-2007
Cytological Effects
72-1501
Enzyme activity
72-1501
Residues/Food and Feed
72-1666
Residues/Soil
72-1662
Residues/Water
72-1662

Treflan
see Trifluralin
Trichlorfon
see also
Organophosphates
Blood/Body Fluids
72-1946
Cardiovascular System
72-1456
Cytological Effects
72-1457
Digestive System
72-1457 72-1946
Endocrine System
72-1946
Enzyme activity
72-1456
Excretory System
72-1456 72-1457
72-1946
Nervous System
72-1456 72-1457
Residues/Plants
72-1651
Residues/Soil
72-1651
Respiratory System
72-1456 72-1457
Toxicity/Experimental
Animals
72-1946
Toxicity/Humans
72-1456 72-1457
Treatment of Poisoning
72-1456 72-1457
2,3,6-Trichlorobenzoate
see 2,3,6-TBA
alpha-Trichloromethyl-p-ethoxybenzyl-p-ethoxyaniline
see also Organochlorines
Metabolism
72-1980
Tri-o-cresyl phosphate
see also Fungicides,
Organophosphates
Analysis
72-1807
Enzyme activity
72-1485 72-1932
Factors Influencing
Metabolism/Toxicity
72-1485
Metabolism
72-1485
Nervous System
72-1515 72-1697
72-1932
Toxicity/Experimental
Animals
72-1485 72-1515
72-1932
Toxicity/Humans
72-1697
Tricyclohexyltin hydroxide
see Plictran
Tri(dimethyldithiocarbamoy
see TTCA
Triethylphosphate
see also
Organophosphates
Factors Influencing
Metabolism/Toxicity
72-1476
Metabolism
72-1476
Toxicity/Experimental
Animals
72-1476

Trifluralin
see also Herbicides
Analysis
72-1562 72-1580
72-1866
Biochemical Effects
72-1966
Endocrine System
72-1966
Prevention
72-1966
Residues/Soil
72-1580 72-1663
72-1858 72-1866
Toxicity/Experimental
Animals
72-1966
Tri-o-tolyl phosphate
see Tri-o-cresyl
phosphate
TTCA
see also Arsenicals
Residues/Food and Feed
72-1412
Vamidothion
see also
Organophosphates
Metabolism
72-1960
Vanalate
see also Carbamates
Residues/Food and Feed
72-1411
Vapam
see Metham
VCS-438
see also Herbicides
Metabolism
72-1497
Warfarin
see also Rodenticides
Factors Influencing
Metabolism/Toxicity
72-1744
Toxicity/Experimental
Animals
72-1952
Weedex
see Simazine
Weedol
see Paraquat
Yalan
see Molinate
Zinc phosphide
see also Rodenticides
Reproduction/Growth
72-1750
Toxicity/Experimental
Animals
72-1750
Zineb
see also Carbamates
Analysis
72-2009
Blood/Body Fluids
72-1946
Digestive System
72-1946
Endocrine System
72-1946
Excretory System
72-1946
Toxicity/Experimental
Animals
72-1946
Z-50
see Ronnel
Z-7272
Toxicity/Experimental
Animals
72-1931

Alternative Controls
72-1356 72-1358
72-1369 72-1585
72-1587 72-1589
72-1590 72-1597
72-1610 72-1611
72-1612 72-1812
72-1816 72-1819
72-1821 72-1825
DDT
72-1364
Analysis
General
BHC isomers
72-1552
DDT
72-1784
EPN
72-1552
Malathion
72-1779
Methyl bromide
72-2010
Organophosphates
72-1798
Bioassay
Carbamates
72-1547
DDT
72-1791
Dicamba
72-1954
Diphenamid
72-2026
Diquat
72-1547
Diuron
72-1800
Herbicides
72-1546 72-1547
72-1632 72-2018
Lindane
72-1791
Mercurials
72-1788 72-2021
Paraquat
72-1547
Picloram
72-2027
Polyoxin D
72-1551
2,3,6-TBA
72-1954
Calorimetry
Carbamates
72-2015
Herbicides
72-2015
Mercurials
72-2015
Organochlorines
72-2015
Organophosphates
72-2015
Cholinesterase/enzyme
72-1565
DDT
72-1575 72-1780
Lindane
72-1575
PCP
72-1575
Chromatography
General
Fluoroacetamide
72-1967
MNPA
72-1967
Polychlorinated
biphenyls
72-2017
Column
Carbaryl
72-1564
Polychlorinated
biphenyls
72-1782
Gas-liquid
Aldrin
72-1553 72-1781
72-1810
Benefin
72-1580
BHC isomers
72-1553
Carbamates
72-2030
Carbaryl
72-1568
Chlorinated
naphthalenes
72-1561
Chlorthiamid
72-1399
Crufomate
72-2016
DDT
72-1548 72-1780
72-1781 72-2013
DDT derived
compounds
72-1781
DDT isomers
72-1553
Dichlobenil
72-1399
5,6-Dichloro-2-
trifluoromethylbenzi
72-1571
Dichlorvos
72-1554
Dicrotophos
72-1554
Dieldrin
72-1553 72-1781
72-1810 72-2035
3,4-Dimethylphenyl
N-methylcarbamate
72-1549
Dioxins
72-1416 72-1572
2,4-D
72-1569
Endrin
72-1553 72-1555
72-1781 72-1783
72-2013
Endrin derived
compounds
72-1555 72-1783
Fenazaflor
72-1571 72-2032
72-2033
Fenitrothion
72-1789
Fluenethyl
72-2031
Fungicides
72-1566
Heptachlor
72-1781 72-1810
Heptachlor epoxide
72-1781 72-1810
Herbicides
72-1566 72-1572
Iodofenphos
72-2037
Iodofenphos derived
compounds
72-2037
Lindane
72-1781 72-1810
Maneb
72-2009
Methomyl
72-2036
Methyl bromide
72-2034
Methyl parathion
72-1789
Mevinphos
72-1554
Monocrotophos
72-1554
Nemacide
72-1508
Organochlorines
72-1416 72-1555
72-1557 72-1558
72-1559 72-1561
72-1567 72-1574
72-1581 72-1633
72-1777 72-1785
72-1787 72-1790
72-1806 72-2014
72-2023 72-2024
Organophosphates
72-1556 72-1566
72-1579 72-1790
Parathion
72-1384 72-1554

PCNB
72-1786
Polychlorinated biphenyls
72-1416 72-1563
72-1639 72-1782
72-1785 72-1796
72-2011 72-2028
72-2029
Polychlorobenzofuran
72-1416
TDE
72-1781 72-2013
Trifluralin
72-1580 72-1866
Zineb
72-2009
Ion-exchange
72-1802
Paper
Bromoxynil
72-1398
Malathion
72-1544
Mercurials
72-1788 72-1803
72-2020 72-2021
Organochlorines
72-2020
Organophosphates
72-2020
Thin-layer
Azinphosmethyl
72-1794 72-2006
Bromoxynil
72-1398
Carbamates
72-1547
Chloramphenicol
72-1804
DDT
72-1575
Demephion
72-2019
Dimethoate
72-1577 72-1794
72-2006
Diquat
72-1547
Ethion
72-2006
Herbicides
72-1547
Landrin
72-1573
Lindane
72-1575
Malathion
72-2006
Methiocarb
72-1573
Organochlorines
72-1574 72-1804
72-2014
Paraquat
72-1547
PCP
72-1575
Phorate
72-2006
Prometryne
72-2006
Propoxor
72-1573
Rotenone
72-1808
Electrometry
Amperometry
Organochlorines
72-1799
Coulometry
Methomyl
72-2036
Polarography
Pentachlorobenzaldox
72-1801
Gravimetry
72-1802
Neutron activation
Aluminum phosphide
72-1624
Spectrometry
General
Carbamates
72-1578
Carbaryl
72-1981
Diazinon
72-1709
Metallic compounds
72-1578
Organophosphates
72-1578
Atomic absorption/ emission
Arsenicals
72-1576
Colorimetry (visible)
Carbamates
72-1797
Carbaryl
72-1564 72-2022
Chlorbromuron
72-2025
DDT
72-1792
Dichlorvos
72-1545
Flucmeturon
72-2025
Fluoroacetamide
72-1967
Malathion
72-1544
Menazon
72-1582
Methyl mercaptophos
72-1805
Metobromuron
72-2025
MNFA
72-1967
Toxaphene
72-2007
Tri-o-cresyl phosphate
72-1807
Fluorometry
Azinphosmethyl
72-2006
Carbaryl
72-1550 72-1570
Dimethoate
72-2006
Ethion
72-2006
Malathion
72-2006
Phorate
72-2006
Prometryne
72-2006
Infrared
Dichlorvos
72-1778
Malathion
72-1778
Polychlorinated biphenyls
72-2011
Schradan
72-1778
Mass spectrometry
Bromacil
72-1562
Bromoxynil
72-1562
Chlordecone
72-1560
Crufomate
72-2016
2,4-D
72-1563
Mirex
72-1560
Parathion
72-1384
Polychlorinated biphenyls
72-2008 72-2017
Trifluralin
72-1562
Neutron activation
72-1802
Polychlorinated biphenyls
72-2011
NMR
Aldrin
72-1793
BHC
72-1583

Dieldrin
72-1793
Endrin
72-1793
Isodrin
72-1793
Radiometry
Organochlorines
72-1556
Organophosphates
72-1556
UV
Bromoxynil
72-1398
Carbamates
72-1635
Parathion
72-1795
Titration
Amitrole
72-2012

Beneficial Effects
72-1360 72-1362
72-1368
Aldrin
72-1367
DDT
72-1367 72-1512
72-1584
Endrin
72-1367
Biochemical Effects
GA-3
72-1760
General
Animals/Experimental
Dieldrin
72-1466
PCP
72-1463
Thanite
72-1945
In vitro
PCP
72-1463
Microorganisms
Paraquat
72-1467
Amino acids/peptides/ proteins
Animals/Experimental
Malathion
72-1532
Ronnel
72-1762
TDE
72-1722
Human
Organophosphates
72-1682
Microorganisms
Aldrin
72-1521
Bandane
72-1521
Chlordane
72-1521
DDT
72-1521
Dieldrin
72-1521
Endrin
72-1521
Heptachlor
72-1521
Lindane
72-1521
Telodrin
72-1521
Carbohydrates
Animals/Experimental
DDT
72-1910
Dicamba
72-1954
Erbon
72-1966
Flucmeturon
72-1966
Fluoroacetamide
72-1968
Linuron
72-1966
MNPA
72-1968
2,3,6-TBA
72-1954
Trifluralin
72-1966
Human
Aldrin
72-1878
Dieldrin
72-1878
Endrin
72-1878
Parathion
72-1437
Microorganisms
Chlordane
72-1523

Catecholamines
Animals/Experimental
DDT
72-1471
DDT isomers
72-1471

Electrolytes
In vitro
DDT
72-1775
Plant
DDT
72-1751

Electron transport
In vitro
DDT
72-1915 72-1916
DDT derived compounds
72-1915
Malathion
72-1731
Parathion
72-1731
Lipids/steroids/sterols
TDE
72-1767
Animals/Experimental
Chlormequat chloride
72-1768
DDT
72-1891
2,4-D
72-1768
TDE
72-1538
Human
DDT
72-1696
In vitro
Amo 1618
72-1764
Phosfon
72-1764
Nucleic Acids
Animals/Experimental
DFP
72-1929
Soman
72-1929
Microorganisms
Aldrin
72-1521
Bandane
72-1521
Chlordane
72-1521 72-1522
DDT
72-1521
Dieldrin
72-1521
Endrin
72-1521
Heptachlor
72-1521
Lindane
72-1521
Telodrin
72-1521

Vitamins/coenzymes
Animals/Experimental
Chlormequat chloride
72-1768
2,4-D
72-1768

Human
Dichlorvos
72-1689

Organophosphates
72-1689
Blood/Body Fluids
General
Animals/Experimental
Dieldrin
72-1466
Blood-brain barrier
Animals/Experimental
Organophosphates
72-1721
Trichlorfon
72-1946
Zineb
72-1946
Cells
Animals/Experimental
Allethrin
72-1933
Carbaryl
72-1964
DDT
72-1964
Methyl mercaptophos
72-1542
Parathion
72-1964
Piperonyl butoxide
72-1933
Pyrethrins
72-1933
In vitro
Sarin
72-1975
Plasma/serum
Animals/Experimental
Allethrin
72-1933
DDT
72-1710 72-1757
72-1891
Di-allate
72-1922
Dioxins
72-1535
Fluoroacetamide
72-1968
MNFA
72-1968
Piperonyl butoxide
72-1933
Pyrethrins
72-1933
Ronnel
72-1762
Thanite
72-1945
Human
DDT
72-1696
Carcinogenesis
General
DDT
72-1364
Animals/experimental
DDT
72-1481 72-1926
Organochlorines
72-1926
TDE
72-1481
Cardiovascular System
General
Animals/Experimental
DDT
72-1710
Disulfiram
72-1462
Sodium
diethyldithiocarbama
72-1462
Human
DDT
72-1696
Blood pressure
Animals/Experimental
Dichlorvos
72-1906
Mevinphos
72-1906
Heart
Animals/Experimental
DDT derived
compounds
72-1911
Dieldrin
72-1911
Dioxins
72-1536
MNFA
72-1969
Human
MNFA
72-1969
Organophosphates
72-1898
Trichlorfon
72-1456
Conferences
72-1358
Cytological Effects
General
DDT
72-1475
Dieldrin
72-1475
Animals/Experimental
Dieldrin
72-1902
Heptachlor
72-1539 72-1737
Human
Trichlorfon
72-1457
In vitro
2,4-D
72-1530
Microsomes
Animals/Experimental
Piperonyl butoxide
72-1470
In vitro
Parathion
72-1924
Piperonyl butoxide
72-1994
Mitochondria
Animals/Experimental
DDT
72-1501
Dieldrin
72-1501
Toxaphene
72-1501
In vitro
DDT
72-1916
Digestive System
General
Animals/Experimental
Barban
72-1474
Heptachlor
72-1737
Paraquat
72-1992
Z-7272
72-1931
Human
Organophosphates
72-1898
Paraquat
72-1446
Trichlorfon
72-1457
Alimentary tract
Animals/Experimental
BHC
72-1486
Copper sulfate
72-1543
DDT
72-1543
Dieldrin
72-1486
Human
Mercurials
72-1440
Parathion
72-1437
Liver
Animals/Experimental
Chlormequat chloride
72-1763
DDT
72-1475 72-1493
72-1526 72-1732
72-1757 72-1758
72-1910
DFP
72-1929

Dieldrin
72-1475 72-1493
72-1758 72-1902
Dioxins
72-1536
Diquat
72-1770
2,4-D
72-1768 72-1950
Endrin
72-1732
Fluoroacetamide
72-1968
Heptachlor
72-1464 72-1539
Lindane
72-1732
Malathion
72-1532
Mercurials
72-1464
MNFA
72-1968
Paraquat
72-1770
Parathion
72-1468
Picloram
72-1950
Pyrethrins
72-1498
Soman
72-1929
Trichlorfon
72-1946
Zineb
72-1946
Human
Aldrin
72-1878
Dieldrin
72-1878
Endrin
72-1878
In vitro
Heptachlor epoxide
72-1934
Parathion
72-1909
Economics
72-1358 72-1952
72-1604
Education/Training
72-1354
Endocrine System
General
Animals/Experimental
Trichlorfon
72-1946
Zineb
72-1946
Adrenal
Animals/Experimental
Dieldrin
72-1466
TDE
72-1538
Z-7272
72-1931
Spleen
Animals/Experimental
Heptachlor
72-1737
Thymus
Animals/Experimental
Dioxins
72-1536
TDE
72-1976
Thyroid
Animals/Experimental
DDT
72-1758 72-1942
DDT derived compounds
72-1911 72-1942
Dieldrin
72-1758 72-1911
Erbon
72-1966
ETU
72-2005
Fluometuron
72-1966
Linuron
72-1966
Maneb
72-2005
Trifluralin
72-1966
Environmental Pollution
72-1360 72-1365
72-1370 72-1586
72-1589 72-1596
72-1597 72-1599
72-1614 72-1811
72-1812 72-1813
72-1816 72-1817
72-1818 72-1820
72-1824 72-1827
72-1830
Aldrin
72-1367
Arsenicals
72-1606
Captan
72-1355
Carbamates
72-1843
Chemosterilants
72-1355
DDT
72-1355 72-1357
72-1361 72-1364
72-1367 72-1373
72-1584 72-1594
72-1603
Dioxins
72-1355 72-1608
Endrin
72-1367
Herbicides
72-1366
Mercurials
72-1605 72-1609
72-1828
Mirex
72-1355
Organochlorines
72-1588 72-1601
72-1605 72-1613
72-1822 72-1828
Organophosphates
72-1361 72-1605
72-1843
Parathion
72-1603
Picloram
72-1829
Polychlorinated biphenyls
72-1355
2,4,5-T
72-1608 72-1829
Enzyme activity
General
Aldrin
72-1521 72-1878
Bandane
72-1521
Biphenyl
72-1738
Carbaryl
72-1964
Chlordane
72-1521 72-1522
72-1523
Chlormequat chloride
72-1768
2,4-D derived compounds
72-1973
DDT
72-1521 72-1526
72-1757 72-1891
72-1910 72-1916
72-1964
DFP
72-1929
Dicamba
72-1954
Dieldrin
72-1521 72-1878
Dioxins
72-1535
Diquat
72-1770
Dyfonate
72-1947

2,4-D
72-1768 72-1973
Endrin
72-1521 72-1878
Heptachlor
72-1521
Lindane
72-1521
Paraquat
72-1770
Parathion
72-1468 72-1964
Piperonyl butoxide
72-1994
Pyrethrins
72-1498
Soman
72-1929
2,3,6-TBA
72-1954
TDE
72-1767
Telodrin
72-1521
Adenosine triphosphatase
DDT
72-1751
Polychlorinated biphenyls
72-1488 72-1978
Aldolase
Aldrin
72-1480
Dieldrin
72-1480
Alkaline phosphatase
Carbaryl
72-1527
DDT
72-1527
Dichlorvos
72-1527
Fenitrothion
72-1527
Aniline hydroxylase
DDT
72-1500
Aromatic N-deacetylase
3-Chloro-p-toluidine
72-1979
Choline acetylase
TDE
72-1976
Cholinesterase
72-1687 72-1704
Amiton
72-1511
Azinphosmethyl
72-1485
Carbamates
72-1499
Carbaryl
72-1527
Coumaphos
72-1485
DDT
72-1481 72-1527
DFP
72-1477 72-1485
72-1514 72-1524
72-1766 72-1977
Di-allate
72-1922
Dichlorvos
72-1527 72-1689
72-1906
Dioxathion
72-1519
Dursban
72-1728
EPN
72-1485
Ethylmethylfluorothi
72-1528
Fenitrothion
72-1527 72-1730
Fenthion
72-1730
Isopropylmethylfluor
72-1528
Malaoxon
72-1511
Malathion
72-1445
Mercurials
72-1693
Mevinphos
72-1906
Mipafox
72-1718
Nemacide
72-1508
Organochlorines
72-1693
Organophosphates
72-1531 72-1682
72-1689 72-1693
72-1706 72-1721
72-1765 72-1920
72-1957
Paraoxon
72-1485 72-1977
Parathion
72-1437 72-1485
72-1677 72-1699
Phosmet
72-1519
Sarin
72-1974 72-1975
72-1996
Soman
72-1953
S 4087
72-1944
TDE
72-1481
TEPP
72-1477 72-1485
Trichlorfon
72-1456
Tri-o-cresyl phosphate
72-1485 72-1932
Z-7272
72-1931
Corticosteroid hydroxylase
TDE
72-1538
DDT-dehydrochlorinase
DDT
72-1465
delta-Aminolevulinic acid synthetase
Lindane
72-1774
Glutamic oxaloacetate transaminase
Carbaryl
72-1527
DDT
72-1527
Dichlorvos
72-1527
Fenitrothion
72-1527
Glutamic pyruvic transaminase
Aldrin
72-1480
Carbaryl
72-1527
DDT
72-1527
Dichlorvos
72-1527
Dieldrin
72-1480
Fenitrothion
72-1527
Malathion
72-1532
Hydroxycorticosteroid dehydrogenase
TDE
72-1538
Lactic dehydrogenase
Heptachlor
72-1464
Mercurials
72-1464
Mixed function oxidases
Aldrin
72-1484
DDT
72-1493
Dieldrin
72-1484 72-1493
Methyl parathion
72-1999

Parathion
72-1484 72-1909
72-1939
Rotenone
72-1719
Sesamex
72-1484
N-Demethylase
DDT
72-1500
Nitroreductase
Parathion
72-1951
Ornithine-carbamyl transferase
Carbaryl
72-1527
DDT
72-1527
Dichlorvos
72-1527
Fenitrothion
72-1527
Peroxidase
Herbicides
72-1502
Succinic dehydrogenase
DDT
72-1501
Dieldrin
72-1501
Heptachlor epoxide
72-1934
Toxaphene
72-1501
Transaminase
MEMC
72-1459
Excretory System
General
Animals/Experimental
Z-7272
72-1931
Human
Endrin
72-1683
Trichlorfon
72-1457
Bladder/ducts
Human
Trichlorfon
72-1456
In vitro
Soman
72-1953
Kidney
Animals/Experimental
BHC
72-1486
DDT
72-1732 72-1910
Dieldrin
72-1486 72-1902
Diquat
72-1770
Endrin
72-1732
Heptachlor
72-1737
Lindane
72-1732
MEMC
72-1459
Paraquat
72-1770
Trichlorfon
72-1946
Zineb
72-1946
Experimental Design
72-1371 72-1611
72-1612 72-1972
DDT
72-1357
Factors Influencing Metabolism/Toxicity
General
DDT
72-1615
DDT derived compounds
72-1615
Dieldrin
72-1615
Dursban
72-1728
Fenitrothion
72-1734
Organochlorines
72-1454
Parathion
72-1454
Polychlorinated biphenyls
72-1615
TDE
72-1615
Adaptation
Warfarin
72-1744
Age
Amitrole
72-1720
Ethephon
72-1903 72-1914
Organochlorines
72-1674
Triethylphosphate
72-1476
Biological magnification
DDT
72-1645
DDT derived compounds
72-1645
Dieldrin
72-1725
TDE
72-1645
Disease state
DDT
72-1711
Dioxathion
72-1519
Hempa
72-1743
Phosmet
72-1519
Humidity
Chloropicrin
72-1458
Interactions
Amitrole
72-1720
Azinphosmethyl
72-1485
BHC isomers
72-1988
Biphenyl
72-1738
Carbaryl
72-1964
Chlordane
72-1708
Coumaphos
72-1485
DDT
72-1435 72-1475
72-1493 72-1526
72-1713 72-1907
72-1910 72-1912
72-1964
DDT derived compounds
72-1435
DFP
72-1477 72-1485
72-1514 72-1524
72-1729
Dieldrin
72-1475 72-1493
72-1902 72-1998
Disulfiram
72-1462
EPN
72-1485
Heptachlor
72-1464
Lindane
72-1988
Mercurials
72-1464
Methoxychlor
72-1708
Paraoxon
72-1485
Parathion
72-1437 72-1485
72-1964
Piperonyl butoxide
72-1736 72-1938

Polychlorinated biphenyls 72-1708
Rotenone 72-1719
Sodium diethyldithiocarbamate 72-1462
TEPP 72-1477 72-1485
Tri-o-cresyl phosphate 72-1485
Light/radiation
Benomyl 72-1889
BHC 72-1759
Pyrethrins 72-1628
Nutritional state
Aldrin 72-1963
BHC isomers 72-1988
Captan 72-1513
Carbaryl 72-1496 72-1513
Chlorpropham 72-1513
DDT 72-1496 72-1513 72-1741 72-1772 72-1845 72-1963
DDT derived compounds 72-1845
Diazinon 72-1513
Dieldrin 72-1845
Endosulfan 72-1513
Lindane 72-1513 72-1988
Malathion 72-1513
Monuron 72-1513
Parathion 72-1496
pH
Sarin 72-1975
Season
DDT 72-1845
DDT derived compounds 72-1845
Dieldrin 72-1845
Sex
Di-allate 72-1922
Heptachlor 72-1737
Organochlorines 72-1674
Species
3-Chloro-p-toluidine 72-1979
DDT 72-1714 72-1755
Temperature
Chloropicrin 72-1458
Immunology
Animals/experimental
BHC 72-1771
Methyl mercaptophos 72-1771
Pyrethrins 72-1936
Humans 72-1702
Benomyl 72-1889
BHC 72-1771
Methyl mercaptophos 72-1771
Integument
Feathers
Animals/Experimental
DDT 72-1449
Mercurials 72-1449
Polychlorinated biphenyls 72-1449
Hair/fur
Animals/Experimental
Di-allate 72-1922
Nails/claws/horn
Animals/Experimental
Amitrole 72-1720
Skin
Animals/Experimental
Pyrethrins 72-1936
Human 72-1702
Benomyl 72-1889
Captafol 72-1684 72-1686
Organochlorines 72-1451
Laws and Regulations
General 72-1599 72-1814
Finland 72-1363
France 72-1591
Germany (BRD) 72-1604
Organochlorines 72-1600
Germany (DDR) 72-1820
Hungary 72-1371
Israel
Amitrole 72-1354
Ammonium Thiocyanate 72-1354
Dalapon 72-1354
Diquat 72-1354
2,4-D 72-1354
Malathion 72-1354
Paraquat 72-1354
Picloram 72-1354
2,4,5-T 72-1354
Japan 72-1830
Sweden
DDT 72-1359
Mercurials 72-1359
USA
General 72-1362
Polychlorinated biphenyls 72-1607
EPA
DDT 72-1823 72-1826
States 72-1813
USSR 72-1372
Metabolism
General
General
Carbamates 72-1965
DDT 72-1739
DDT derived compounds 72-1739
Organochlorines 72-1965

Organophosphates
72-1461
Biotransformation
72-1949
Carbamates
72-1948 72-1958
DDT
72-1714 72-1948
Fungicides
72-1948
Organochlorines
72-1958
Organophosphates
72-1840 72-1948
72-1958
Distribution/Storage
DDT
72-1475
Dieldrin
72-1475
Bat
Biotransformation
DDT
72-1845
DDT derived
compounds
72-1845
Dieldrin
72-1845
Distribution/Storage
DDT
72-1845
DDT derived
compounds
72-1845
Dieldrin
72-1845
Birds
Biotransformation
3-Chloro-p-toluidine
72-1979
Chicken
Carbaryl
72-1507
General
DDT
72-1941
DDT derived
compounds
72-1941
DDT isomers
72-1941
Biotransformation
Propham
72-1872
Distribution/Storage
DDT
72-1772
Nemacide
72-1508
Cow
General
Carbaryl
72-1981
Absorption
R-3828
72-1517
Distribution/Storage
Ceresan L
72-1473
Excretion
DDT
72-1487
DDT derived
compounds
72-1487
Fenitrothion
72-1516
R-3828
72-1517
Dog
Distribution/Storage
Carbaryl
72-1541
Fish
General
2,4-D
72-1955
Absorption
Aldrin
72-1908
DDT
72-1908
Dieldrin
72-1908
Endrin
72-1908
Biotransformation
DDT
72-1776
DDT derived
compounds
72-1776
Polychlorinated
biphenyls
72-1776
Guinea pig
Distribution/Storage
Dieldrin
72-1902
Human
General
72-1704
Absorption
Aldrin
72-1935
Carbaryl
72-1935
Dieldrin
72-1935
Lindane
72-1935
Malathion
72-1935
Parathion
72-1935
Pralidoxime
72-1925
Biotransformation
Parathion
72-1795
Distribution/Storage
DDT
72-1756
Dieldrin
72-1756
In vitro
Biotransformation
Biphenyl
72-1738
Carbaryl
72-1997
DDT
72-1912
DFP
72-1524
Diquat
72-1717
Dyfonate
72-1947
Fluorodifen
72-1983
Methyl parathion
72-1999
Parathion
72-1951 72-1999
Piperonyl butoxide
72-1742
Distribution/Storage
2,4-DB
72-2001
2,4-D
72-2001
MCPA
72-2001
MCPB
72-2001
2,4,5-TB
72-2001
2,4,5-T
72-2001
Insects
Biotransformation
Chlorbicyclen
72-1530
Endosulfan
72-1530
1,4,5,6,7,7-
Hexachloro-
norbornene-2,3-bis-c
72-1530
Polychlorinated
biphenyls
72-1616
alpha-
Trichloromethyl-p-
ethoxybenzyl-p-ethox
72-1980
Microorganisms
Absorption
BHC
72-1759

DDT
72-1915
DDT derived
compounds
72-1915
Biotransformation
Aldrin
72-1962
Barban
72-1752
Carbamates
72-1923
Carbaryl
72-1478
DDT
72-1713 72-1915
72-1962 72-1982
DDT derived
compounds
72-1915
Diazinon
72-1504
Dicamba
72-1865
Dichlobenil
72-1864
Dichlorprop
72-1865
Dieldrin
72-1397 72-1913
72-1962
Endosulfan
72-1913
Endrin
72-1913 72-1962
Fenoprop
72-1865
Herbicides
72-1923
Malathion
72-1469
MCCP
72-1865
MCPA
72-1865
MCPB
72-1865
Mobam
72-1707
PCP
72-1495 72-1959
Propanil derived
compounds
72-1984
2,3,6-TBA
72-1617
Mouse
General
DDT
72-1741 72-1755
Dieldrin
72-1998
Absorption
BHC isomers
72-1989
Lindane
72-1989
Biotransformation
Azinphosmethyl
72-1485
Coumaphos
72-1485
DFP
72-1485
EPN
72-1485
Organophosphates
72-1920
Paraoxon
72-1485
Parathion
72-1485
TEPP
72-1485
alpha-
Trichloromethyl-p-
ethoxybenzyl-p-ethox
72-1980
Tri-o-cresyl
phosphate
72-1485
Excretion
BHC isomers
72-1989
Lindane
72-1989
Pigeon
Distribution/Storage
DDT derived
compounds
72-1911
Dieldrin
72-1911
Plants
General
Aldrin
72-1408 72-1431
Chlordane
72-1715
Dieldrin
72-1408 72-1431
Ethephon
72-1903 72-1914
Formothion
72-1503
Heptachlor
72-1431
Malaoxon
72-1724
Malathion
72-1724
Organophosphates
72-1961
VCS-438
72-1497
Absorption
Aldrin
72-1405
Arsenicals
72-1400
BHC isomers
72-1405
DDT
72-1405
Endrin
72-1405
Heptachlor
72-1405
Lead
72-1400
Biotransformation
Atrazine
72-1956
Heptachlor
72-1716
Simazine
72-1956
Thiophanate
72-1921
Vamidothion
72-1960
Distribution/Storage
Aldrin
72-1401 72-1408
BHC isomers
72-1401
DDT
72-1848
DDT derived
compounds
72-1401
DDT isomers
72-1401
Dieldrin
72-1401 72-1408
Endrin
72-1401
Ethephon
72-1472
Heptachlor
72-1401
Heptachlor epoxide
72-1401
Mercurials
72-1846
Vamidothion
72-1960
Rabbit
Biotransformation
Ethylmethylfluorothi
72-1528
Isopropylmethylfluor
72-1528
Parathion
72-1909
Distribution/Storage
BHC isomers
72-1988
Fluoroacetamide
72-1967
Lindane
72-1988

MNPA
72-1967
Rat
General
Aldrin
72-1963
BHC isomers
72-1773
Chlorpropham
72-1723
DDT
72-1963
Granosan
72-1769
Mercurials
72-1712
Triethylphosphate
72-1476
Absorption
Carbaryl
72-1964
DDT
72-1964
Parathion
72-1964
Biotransformation
Atrazine
72-1506
Carbaryl
72-1997
C-2307
72-1753
DDT
72-1489
DDT isomers
72-1489
2,6-Di-tert-butyl-4-nitrophenol
72-2004
Dicrotophos
72-1752
Dieldrin
72-1505
Ethylmethylfluorothi
72-1528
Isopropylmethylfluor
72-1528
Lindane
72-1490
Parathion
72-1468
Pyrethrins
72-1735
Distribution/Storage
BHC isomers
72-1987 72-1988
Chlordane
72-1708
DDT
72-1711
Diquat
72-2000
Lindane
72-1988
Methoxychlor
72-1708
Paraquat
72-2000
Polychlorinated biphenyls
72-1708 72-1782
Excretion
2,6-Di-tert-butyl-4-nitrophenol
72-2004
Polychlorinated biphenyls
72-1782
Pyrethrins
72-1735
Scud.
Absorption
Polychlorinated biphenyls
72-1616
Sheep
Biotransformation
Diazinon
72-1709
Excretion
DDT
72-1873
Turkey
Absorption
DDT
72-1853
Morbidity and Mortality Statistics
Czechoslovakia
72-1702
Japan
72-1685 72-1687
Captafol
72-1686
Mercurials
72-1693
Organochlorines
72-1681 72-1693
Organophosphates
72-1444 72-1681
72-1693
Puerto Rico
Parathion
72-1881
Rumania
Organochlorines
72-1454
Parathion
72-1454
Singapore
Organophosphates
72-1900
Switzerland
Rodenticides
72-1455
USA
DDT
72-1891
Musculoskeletal System
General
Human
Endrin
72-1683
Diaphragm
In vitro
DFP
72-1977
Paraoxon
72-1977
Muscle, smooth
In vitro
Soman
72-1953
Muscle, striated
Human
DFP
72-1441
Parathion
72-1437
In vitro
Polychlorinated biphenyls
72-1978
Skeleton/bone
Animals/Experimental
Amitrole
72-1720
Dioxins
72-2003
2,4-D
72-1995
2,4,5-T
72-1995 72-2003
Mutagenesis/Teratogenesis
General
72-1370 72-1815
72-1972
Animals/experimental
72-1673
Amitrole
72-1720
Apholate
72-1971
Carbaryl
72-1993
Chlormequat chloride
72-1971
Dioxins
72-1745 72-2003
2,4,-D
72-1995
Heptachlor
72-1464
Malathion
72-1971
Mercurials
72-1464
PMA
72-1971
Sodium arsenate
72-1971

Sodium arsenite
72-1494
Thio-TEPA
72-1971
2,4,5-T
72-1745 72-1995
72-2003
Humans
72-1679
Captan
72-1890
Maleic hydrazide
72-1890
2,4,5-T
72-1678
In vitro
2,4-D
72-1534
Microorganisms
Bensulide
72-1510
Herbicides
72-1509
Wildlife
DDT
72-1449
Mercurials
72-1449
Polychlorinated
biphenyls
72-1449
Nervous System
General
Animals/Experimental
Ceresan L
72-1473
DDT
72-1471 72-1907
DDT isomers
72-1471
2,4-D
72-1950
Organophosphates
72-1525 72-1920
Picloram
72-1950
Human
72-1704 72-1885
Endrin
72-1439
Mercurials
72-1440
Organochlorines
72-1442
Organophosphates
72-1442 72-1703
72-1898
Parathion
72-1437
PCP
72-1439
N-
Tetrachloroethylthio
72-1439
Thallium
72-1436 72-1884
In vitro
Organophosphates
72-1765 72-1957
Behavior
Animals/Experimental
Fenitrothion
72-1985
Brain
Animals/Experimental
DDT
72-1758
Dicamba
72-1954
Dieldrin
72-1758 72-1902
GA-3
72-1760
2,3,6-TBA
72-1954
TDE
72-1722
Human
MNFA
72-1899
Trichlorfon
72-1457
In vitro
Malathion
72-1731
Parathion
72-1731
Sarin
72-1974 72-1996
Demyelination
Animals/Experimental
Tri-o-cresyl
phosphate
72-1515
EEG
MNFA
72-1899
Peripheral nerves
Animals/Experimental
Strychnine
72-1740
Tri-o-cresyl
phosphate
72-1932
Human
DFP
72-1441
Mercurials
72-1880
In vitro
DDT
72-1775
Soman
72-1953
Polyneuritis
Human
Thallium
72-1697
Trichlorfon
72-1456
Tri-o-creysl
phosphate
72-1697
Spinal cord
Animals/Experimental
Strychnine
72-1740
Sympathetic nerves
Animals/Experimental
Dichlorvos
72-1906
Mevinphos
72-1906
In vitro
DFP
72-1729
Nomenclature
72-1595
Photodecomposition
Aldrin
72-1842
Azinphosmethyl
72-1647
Dieldrin
72-1842
m-(N,N-
Dimethylformamidine)
phenyl N-methylcarb
72-1424
Diquat
72-1717
Endosulfan
72-1842
Endrin
72-1555
Endrin derived
compounds
72-1555
Heptachlor
72-1871
Organochlorines
72-1671 72-1777
Proparthrin
72-1834
Rotenone
72-1870

Prevention
General
72-1362
Amitrole
72-1354
Ammonium Thiocyanate
72-1354
Barban
72-1452
Chloropicrin
72-1458
Dalapon
72-1354
Di-allate
72-1452

Dicresyl
72-1452
Diquat
72-1354
2,4-D
72-1354
EPTC
72-1452
Erbon
72-1966
Fluometuron
72-1966
Lindane
72-1888
Linuron
72-1966
Malathion
72-1354
Maneb
72-1452
Molinate
72-1452
Paraquat
72-1354
Picloram
72-1354
Trifluralin
72-1966
2,4,5-T
72-1354
Decontamination
Organophosphates
72-1879
Disposal
Malathion
72-1687
Parathion
72-1887
Poison Control Centers
Switzerland
Rodenticides
72-1455
Protective equipment
Dichlorvos
72-1641
Hinosan
72-1701
Safe packaging
BHC
72-1448
DDT
72-1448
Malathion
72-1445
Menazon
72-1448
Nippon
72-1448
Paraquat
72-1448
Simazine
72-1448
Reproduction/Growth
General
Animals/Experimental
Carbaryl
72-1964 72-1993
DDT
72-1512 72-1940
72-1964
DDT derived
compounds
72-1940
DDT isomers
72-1940
Heptachlor
72-1464
Mercurials
72-1464
Parathion
72-1964
Polychlorinated
biphenyls
72-1491
S 4087
72-1944
In vitro
2,4-D
72-1534
Microorganisms
Calcium cyanamide
72-1927
Metham
72-1761
Methyl parathion
72-1761
Picloram
72-2002
Simazine
72-1927
Sodium ethyl
xanthogenate
72-1761
TCA
72-1927
Wildlife
DDT
72-1449 72-1658
72-1663
Mercurials
72-1449 72-1663
Polychlorinated
biphenyls
72-1449 72-1659
Eggshell effects
Animals/Experimental
DDT
72-1757 72-1758
72-1939
Diazinon
72-1939
Dieldrin
72-1758 72-1943
Lindane
72-1939
Malathion
72-1939
Parathion
72-1943
Wildlife
Chlordane
72-1598
DDT
72-1438 72-1598
72-1659
DDT derived
compounds
72-1438
Dieldrin
72-1438
Mercurials
72-1438
Organochlorines
72-1893
Polychlorinated
biphenyls
72-1598
TDE
72-1438
Embryo/fetus
Animals/Experimental
Dieldrin
72-1902
Sodium arsenite
72-1434
Human
Mercurials
72-1652
Estrogens
Animals/Experimental
DDT
72-1710
Fertility
Animals/Experimental
DDT
72-1533 72-1939
Diazinon
72-1939
Dieldrin
72-1943
Lindane
72-1939
Malathion
72-1939
Parathion
72-1943
Zinc phosphide
72-1750
Wildlife
DDT
72-1438
DDT derived
compounds
72-1438
Dieldrin
72-1438
Mercurials
72-1438
TDE
72-1438

Gametogenesis
Animals/Experimental
BHC
72-1486
Chlormequat chloride
72-1768
Dieldrin
72-1486
2,4-D
72-1768
Growth
Ethephon
72-1472
Animals/Experimental
Di-allate
72-1922
2,4-D
72-1991
Mercurials
72-1712
2,4,5-T
72-1991
Microorganisms
DDT
72-1483
Paraquat
72-1467
Polychlorinated biphenyls
72-1483
Plant
DDT
72-1483
Polychlorinated biphenyls
72-1483
Placental transfer
Animals/Experimental
DDT
72-1741
Mercurials
72-1712
Human
Mercurials
72-1631
Reproductive organs, female
Animals/Experimental
Carbaryl
72-1986
2,4-D derived compounds
72-1973
2,4-D
72-1973
Reproductive organs, male
Animals/Experimental
Carbaryl
72-1986
Endrin
72-1919
Lindane
72-1919

Residue Degradation
General
Aldrin
72-1842
Carbamates
72-1635
Carbofenthion
72-1849
Coumaphos
72-1849
DDT
72-1386
Diazinon
72-1504 72-1849
Dieldrin
72-1842
Endosulfan
72-1842
Fenitrothion
72-1849
Malathion
72-1849
Organochlorines
72-1671
Organophosphates
72-1561
Phorate
72-1849
Proparthrin
72-1834
Animals/domestic
Aldrin
72-1618
DDT
72-1873
Dieldrin
72-1618
Heptachlor
72-1618
Heptachlor epoxide
72-1618
Food and Feed
72-1372
Azinphosmethyl
72-1433
Chlordimeform
72-1421 72-1422
Chlorthiamid
72-1399
DDT
72-1791
Diazinon
72-1433
Dichlobenil
72-1399
Dichlorvos
72-1625
Dieldrin
72-1433
Dimethoate
72-1433
Endrin
72-1433
Ethion
72-1433
Heptachlor
72-1433
Lindane
72-1791
Malathion
72-1387 72-1625
72-1652
Methyl parathion
72-1433
Parathion
72-1433
Soil
72-1372
Alachlor
72-1414
Atrazine
72-1850
Barban
72-1752
Bromoxynil
72-1398
Carbaryl
72-1478
DDT
72-1425 72-1868
DDT derived compounds
72-1425 72-1868
DDT isomers
72-1425
2,6-Di-tert-butyl-4(alpha, alpha-dimethylbenzyl)pheno
72-1928
Dicamba
72-1865
Dichlobenil
72-1864
Dichlorprop
72-1865
Dieldrin
72-1383 72-1397
Fenoprop
72-1865
Flucmeturon
72-1415
Herbicides
72-1502 72-1633
MCCP
72-1865
MCPA
72-1865
MCPB
72-1865
Metham
72-1761
Methyl parathion
72-1761
Parathion
72-1377 72-1648
Propanil derived compounds
72-1423

Simazine
72-1861
Sodium ethyl xanthogenate
72-1761
TDE
72-1868
Water
Azinphosmethyl
72-1647
Carbamates
72-1843
DDT
72-1776 72-1982
DDT derived compounds
72-1776
Dichlorvos
72-1554
Dicrotophos
72-1554
Endosulfan
72-1393
Malathion
72-1469
Mevinphos
72-1554
Monocrotophos
72-1554
Organophosphates
72-1843
Parathion
72-1554
PCP
72-1959
Polychlorinated biphenyls
72-1776
2,3,6-TBA
72-1617

Residue Dynamics
72-1654 72-1821
72-1877
Aldrin
72-1629
Atrazine
72-1850
BHC
72-1638
DDT
72-1425 72-1756
DDT derived compounds
72-1425
DDT isomers
72-1425
Dieldrin
72-1622 72-1629
72-1756
Herbicides
72-1374
Lindane
72-1622

Residue Removal
General
72-1630
Malathion
72-1887
Parathion
72-1887
Food and Feed
72-1862
Aluminum phosphide
72-1388
DDT
72-1839
Phosphine
72-1634
Soil
BHC
72-1638
Water
Endosulfan
72-1393
Lindane
72-1841

Residue/Air
General
DDT
72-1357 72-1429
72-1620 72-1833
DDT derived compounds
72-1429
2,4-D
72-1833
Polychlorinated biphenyls
72-1429
Greenhouse
Aldrin
72-1642
Dichlorvos
72-1641
Industrial
Dichlorvos
72-1689
Organophosphates
72-1689

Residues/Domestic Animals
General
Organochlorines
72-1895
Chicken
Propham
72-1872
Tetrachlorvinphos
72-1727
Cow
Dimethoate
72-1668
Pig
Heptachlor
72-1432
Pigs
Aldrin
72-1618
Dieldrin
72-1618
Heptachlor
72-1618
Heptachlor epoxide
72-1618

Residues/Food and Feed
General
72-1376 72-1630
72-1802
Aldrin
72-1643
Benomyl
72-1411
BHC
72-1836
BHC isomers
72-1643
DDT
72-1643
Dimethoate
72-1668
Fenitrothion
72-1789
Hydroxyisoxazole
72-1411
Iodofenphos
72-2037
Iodofenphos derived compounds
72-2037
Methomyl
72-1410
Methyl parathion
72-1789
NH-6967
72-1411
Nitralin
72-1411
Organochlorines
72-1379
Organophosphates
72-1961
Vanalate
72-1411
Animal feed
BHC
72-1835
DDT
72-1666
Lindane
72-1666
Sodium arsenite
72-1656
Toxaphene
72-1666
Animal/Feed
Heptachlor
72-1432
Apples
Plictran
72-1874

Baby food
DDT
72-1848
Beans
Formothion
72-1503
Malathion
72-1427
Berries
DDT
72-1851
Dichlofluanid
72-1851
Malathion
72-1851
Cabbage
Fenitrothion
72-1409
Cereals
Disulfoton
72-1653
Malathion
72-1652
Organophosphates
72-1840
Corn
Aldrin
72-1618
Dieldrin
72-1618
Fensulfothion
72-1426
Heptachlor
72-1618
Heptachlor epoxide
72-1618
Cucumbers
Arsenicals
72-1400
Lead
72-1400
Dairy products
BHC
72-1835
DDT
72-1420 72-1627
72-1661
Fenitrothion
72-1516 72-1537
Organochlorines
72-1588
Polychlorinated
biphenyls
72-1420
Eggplant
Aldrin
72-1401
BHC isomers
72-1401
DDT derived
compounds
72-1401
DDT isomers
72-1401
Dieldrin
72-1401
Endrin
72-1401
Heptachlor
72-1401
Heptachlor epoxide
72-1401
Eggs
Aldrin
72-1395
DDT
72-1395
Dieldrin
72-1395
Hexachlorobenzene
72-1395
Lindane
72-1395
Tetrachlorvinphos
72-1727
Fish
DDT
72-1839
Fruit
Fenazaflor
72-2032 72-2033
Fluenethyl
72-2031
Fruits
Benomyl
72-1863
Chlordimeform
72-1421 72-1422
Formothion
72-1434
Gooseberries
Chlortbiamid
72-1399
Dichlobenil
72-1399
Grain
Azinphosmethyl
72-1433
Diazinon
72-1433
Dieldrin
72-1433
Dimethoate
72-1433
Endrin
72-1433
Ethion
72-1433
Heptachlor
72-1433
Methyl parathion
72-1433
Organochlorines
72-1557
Parathion
72-1433
Herbs
Lindane
72-1382
Meat
DDT
72-1661
Fenitrothion
72-1537
Organochlorines
72-1646
Oils
DDT
72-1792
Pears
Plictran
72-1874
Potatoes
DNOC
72-1665
2,4-D
72-1655
Methyl bromide
72-1396
Poultry
Organochlorines
72-1619
Rice
BHC isomers
72-1406
Kitazin-P
72-1381
Mercurials
72-1846
Silage
Fenitrothion
72-1516
Strawberries
Simazine
72-1861
TTCA
72-1412
Tea
Arsenicals
72-1576
BHC isomers
72-1402
DDT
72-1402 72-1548
DDT derived
compounds
72-1402
DDT isomers
72-1402
Parathion
72-1384
Tomatoes
Diphenamid
72-1413
Total diet
Aldrin
72-1832
Arsenicals
72-1852
BHC
72-1832
DDT
72-1832

DDT derived compounds 72-1832
Dieldrin 72-1832
Organochlorines 72-1837 72-1852
Organophosphates 72-1852
Turkey
DDT 72-1710
Turnips
Aldrin 72-1408
Dieldrin 72-1408
Vegetables
Aldrin 72-1405
BHC isomers 72-1405
DDT 72-1405 72-1667 72-1848
Demephion 72-2019
Endrin 72-1405
Heptachlor 72-1405
Lindane 72-1667
Wheat
Aluminum phosphide 72-1388
Dichlorvos 72-1625
Dieldrin 72-1428
Malathion 72-1387 72-1625
Phosphine 72-1634

Residues/Humans
General 72-1630 72-1802
DDT 72-1876
Menazon 72-1582
Mercurials 72-1631
Polychlorinated biphenyls 72-1430
Adipose
Aldrin 72-1395
BHC 72-1390
DDT 72-1390 72-1395
DDT derived compounds 72-1390
Dieldrin 72-1395
Hexachlorobenzene 72-1395
Lindane 72-1390 72-1395
Organochlorines 72-1639 72-1674
Polychlorinated biphenyls 72-1639
Blood
Aldrin 72-1878
DDT 72-1435 72-1615 72-1696 72-1891
DDT derived compounds 72-1435 72-1615
Dieldrin 72-1615 72-1878
Endrin 72-1878
Polychlorinated biphenyls 72-1615
TDE 72-1615
Milk
72-1376
Aldrin 72-1660
BHC 72-1380 72-1660
BHC isomers 72-1644
DDT 72-1378 72-1380 72-1660
DDT derived compounds 72-1644
DDT isomers 72-1644
Dieldrin 72-1380 72-1660
Endrin 72-1660
Heptachlor 72-1660
Heptachlor epoxide 72-1378
Lindane 72-1378
Organochlorines 72-1664 72-1875
Organs
BHC 72-1380
DDT 72-1380
Dieldrin 72-1380
Mercurials 72-1838
Urine
DDT 72-1637 72-1891
Parathion 72-1675

Residues/Plants
General
DDT 72-1623
Corn
Aldrin 72-1431
Dieldrin 72-1431
Heptachlor 72-1431
Grass
Chlordane 72-1651
Diazinon 72-1651
Diphenamid 72-1413
Dyfonate 72-1651
Fensulfothion 72-1426
Trichlorfon 72-1651
Maple
Carbaryl 72-1564
Ornamentals
Diphenamid 72-1413
Tobacco
Aldrin 72-1403 72-1404
BHC isomers 72-1403 72-1404
DDT 72-1386 72-1403 72-1404 72-2013
Dieldrin 72-1403 72-1404
Endrin 72-1403 72-2013
TDE 72-2013

Residues/Soil
General 72-1672 72-1802
Benefin 72-1580
BHC isomers 72-1407
Chlordane 72-1651

DDT
72-1636 72-1640
72-1662 72-1667
72-1780 72-1853
Diazinon
72-1651
Dieldrin
72-1383 72-1640
72-1649
Dyfonate
72-1651
Heptachlor epoxide
72-1650
Herbicides
72-1632 72-1633
Lindane
72-1667
Organochlorines
72-1670
Simazine
72-1861
Toxaphene
72-1662
Trichlorfon
72-1651
Trifluralin
72-1580 72-1866

Adsorption
72-1391
Ametryne
72-1855
Atrazine
72-1854 72-1855
BHC
72-1638

Movement
72-1860
Agallol
72-1389
Atrazine
72-1375
Benefin
72-1663
Bensulide
72-1663
Carbamates
72-1547
Diazinon
72-1375
Diquat
72-1547
Dyfonate
72-1867
Herbicides
72-1547 72-2018
Paraquat
72-1547
Picloram
72-1857
Propachlor
72-1375
Trifluralin
72-1663

Volatilization
DDT
72-1620
Dieldrin
72-1856
Trifluralin
72-1858

Residues/Water
General
72-1813 72-1860
Aldrin
72-1643 72-1781
BHC
72-1638
BHC isomers
72-1407 72-1643
Chlordane
72-1385
DDT
72-1385 72-1417
72-1643 72-1662
72-1781
DDT derived
compounds
72-1385 72-1417
72-1781
Dieldrin
72-1385 72-1622
72-1781
Endrin
72-1781
Heptachlor
72-1781
Heptachlor epoxide
72-1781
Herbicides
72-1859
Lindane
72-1385 72-1622
72-1781
TDE
72-1385 72-1781
Toxaphene
72-1662
Estuaries
DDT
72-1640
Dieldrin
72-1640
Groundwater
Agallol
72-1389
Oceans
DDT
72-1621
Organochlorines
72-1613
Rivers
Endosulfan
72-1393
Sludge
Organochlorines
72-1806
Streams
DDT
72-1623

Residues/Wildlife
General
DDT
72-1394 72-1623
DDT derived
compounds
72-1394
Dieldrin
72-1394 72-1443
Polychlorinated
biphenyls
72-2017
TDE
72-1394
Birds
DDT
72-1438 72-1449
72-1657 72-1658
72-1659 72-1831
DDT derived
compounds
72-1438
Dieldrin
72-1438 72-1657
Mercurials
72-1438 72-1449
72-1831 72-1844
Polychlorinated
biphenyls
72-1449 72-1657
72-1831
Polychlorinated
terphenyls
72-1418
TDE
72-1438
Eggs
DDT
72-1438 72-1645
72-1659 72-1847
DDT derived
compounds
72-1438 72-1626
72-1645 72-1847
Dieldrin
72-1438 72-1626
Dioxins
72-1416
Lindane
72-1847
Mercurials
72-1438
Organochlorines
72-1416
Polychlorinated
biphenyls
72-1416
Polychlorinated
terphenyls
72-1418
Polychlorobenzofuran
72-1416

TDE
72-1438 72-1645
Fish/aquatic/marine life
DDT
72-1392 72-1419
72-1449 72-1621
72-1640 72-1673
72-1688 72-1831
72-1839
DDT derived compounds
72-1392 72-1419
Dieldrin
72-1640
Dioxins
72-1416
Mercurials
72-1449 72-1831
Organochlorines
72-1416
Polychlorinated biphenyls
72-1416 72-1419
72-1449 72-1616
72-1831
Polychlorobenzofuran
72-1416
TDE
72-1392
Mammals
DDT
72-1845
DDT derived compounds
72-1845
Dieldrin
72-1845
Respiratory System
General
Animals/Experimental
Captan
72-1513
Carbaryl
72-1513
Chlorpropham
72-1513
DDT
72-1513
Diazinon
72-1513
Diquat
72-1770
Endosulfan
72-1513
Lindane
72-1513
Malathion
72-1513
Monuron
72-1513
Paraquat
72-1770
Human
Trichlorfon
72-1456
Lung
Animals/Experimental
Dieldrin
72-1902
Dioxins
72-1536
Diquat
72-2000
Paraquat
72-1917 72-1918
72-2000
Human
Paraquat
72-1446 72-1897
Parathion
72-1437
Trichlorfon
72-1457
Upper respiratory tract
Animals/Experimental
Methyl mercaptophos
72-1542
Reviews
72-1356 72-1369
72-1370 72-1371
72-1372 72-1391
72-1597 72-1614
72-1630 72-1672
72-1704 72-1815
72-1817 72-1830
72-1949
Apholate
72-1971
Arsenicals
72-1602 72-1606
Carbamates
72-1578 72-1843
72-1948 72-1958
Chlordane
72-1598
Chlormequat chloride
72-1971
DDT
72-1598 72-1714
72-1926 72-1948
Fungicides
72-1948
Herbicides
72-1859
Lead
72-1680
Lindane
72-1888
Malathion
72-1971
Mercurials
72-1605 72-1609
72-1680 72-1828
Metallic compounds
72-1578
Organochlorines
72-1442 72-1605
72-1613 72-1646
72-1828 72-1895
72-1926 72-1958
Organophosphates
72-1442 72-1461
72-1578 72-1605
72-1798 72-1840
72-1843 72-1896
72-1948 72-1958
72-1961
PMA
72-1971
Polychlorinated biphenyls
72-1598 72-2017
Sodium arsenate
72-1971
Sodium chlorate
72-1680
TDE
72-1460
Thio-TEPA
72-1971
2,4,5-T
72-1678
Sensory System
Olfaction
Animals/Experimental
Methyl mercaptophos
72-1542
Vision
Animals/Domestic
Carbamates
72-1695
Organophosphates
72-1695
Animals/Experimental
DFP
72-1514
Fenitrothion
72-1730
Fenthion
72-1730
Organophosphates
72-1531
Human
Dimethoate
72-1930
Malathion
72-1445
Mercurials
72-1693
Organochlorines
72-1451 72-1693
Organophosphates
72-1444 72-1453
72-1531 72-1691
72-1693 72-1703
72-1898
Parathion
72-1437
Therapeutic use
Cancer
DDT
72-1481 72-1594
TDE
72-1481 72-1540

Cushing's Syndrome
TDE
72-1460 72-1529
72-1904
Diagnosis
TDE
72-1905
Infertility
TDE
72-1763
Jaundice
DDT
72-1746
Trichinellosis
Ronnel
72-1762
Tumors
TDE
72-1970
Virus infection
TDE
72-1479
Toxicity/Domestic Animals
General
Organochlorines
72-1895
Organophosphates
72-1896
Rodenticides
72-1455
Chicken
Carbamates
72-1894
Organochlorines
72-1894
Organophosphates
72-1894
Cow
Carbamates
72-1695
Organochlorines
72-1883
Organophosphates
72-1695 72-1883
Toxicity/Experimental Animals
General
72-1591
Barban
72-1474
BAY 94337
72-1748
Carbaryl
72-1749
DDT
72-1749
2,6-Di-tert-butyl-4(alpha, alpha-dimethylbenzyl) pheno
72-1928
Erbon
72-1966
Fenitrothion
72-1537
Flucmeturon
72-1966
Lindane
72-1888
Linuron
72-1966
Organophosphates
72-1461 72-1525
Piperonyl butoxide
72-1736
Trifluralin
72-1966
Bat
DDT
72-1845
DDT derived compounds
72-1845
Dieldrin
72-1845
Birds
72-1990
Fenthion
72-1937
Cat
DFP
72-1514
Strychnine
72-1740
Chicken
Amitrole
72-1720
DDT
72-1758 72-1772
72-1940
DDT derived compounds
72-1940
DDT isomers
72-1940
Dieldrin
72-1758
Dursban
72-1728
2,4-D
72-1991
Nemacide
72-1508
Organophosphates
72-1920
S 4087
72-1944
Tetrachlorvinphos
72-1727
Tri-o-cresyl phosphate
72-1515
2,4,5-T
72-1991
Cow
Ceresan L
72-1473
Dioxathion
72-1519
Fenitrothion
72-1516
Phosmet
72-1519
Crab
Dieldrin
72-1754
Crustaceans
Polychlorinated biphenyls
72-1491
Dog
Allethrin
72-1933
Carbaryl
72-1541
DDT
72-1512 72-1926
Fenitrothion
72-1730
Fenthion
72-1730
Organochlorines
72-1926
Organophosphates
72-1531
Piperonyl butoxide
72-1933
Pyrethrins
72-1933
TDE
72-1538 72-1722
Duck
DDT
72-1493
Dieldrin
72-1493 72-1943
Parathion
72-1943
Fish
Aldrin
72-1908
BHC
72-1486
Carbaryl
72-1986
DDT
72-1776 72-1908
DDT derived compounds
72-1776
Dieldrin
72-1486 72-1908
2,4-D
72-1950
Endrin
72-1908
Fenitrothion
72-1985
PCP
72-1463
Picloram
72-1950

Polychlorinated biphenyls
72-1488 72-1776
72-1978
Rotenone
72-1482 72-1719
Thanite
72-1945
Frog
Copper sulfate
72-1543
DDT
72-1543
Guinea Pig
Carbamates
72-1518
Dieldrin
72-1902
Organophosphates
72-1518
Pyrethrins
72-1936
Loris
Tri-o-cresyl phosphate
72-1932
Microorganisms
Aldrin
72-1521
Bandane
72-1521
Chlordane
72-1521 72-1522
72-1523
DDT
72-1521
Dieldrin
72-1521
Endrin
72-1521
Heptachlor
72-1521
Lindane
72-1521
Telodrin
72-1521
Mouse
Azinphosmethyl
72-1485
Coumaphos
72-1485
DFP
72-1485
EPN
72-1485
Fluoroacetamide
72-1967
MNFA
72-1967
Organophosphates
72-1920
Paraoxon
72-1485
Parathion
72-1485
Piperonyl butoxide
72-1470
TEPP
72-1485
Tri-o-cresyl phosphate
72-1485
Z-7272
72-1931
Ostracods
Aldrin
72-1726
Dieldrin
72-1726
Pheasant
Aldrin
72-1492
Pigeon
DDT derived compounds
72-1911
Dieldrin
72-1911
Quail
DDT
72-1757
Zinc phosphide
72-1750
Rabbit
Carbaryl
72-1527
Chlormequat chloride
72-1768
DDT
72-1527 72-1732
Dicamba
72-1954
Dichlorvos
72-1527
Dimethoate
72-1930
2,4-D
72-1768
Endrin
72-1732
Fenitrothion
72-1527
Fluoroacetamide
72-1968
GA-3
72-1760
Lindane
72-1732
Methyl bromide
72-1747
Methyl mercaptophos
72-1542
MNFA
72-1968 72-1969
Paraquat
72-1992
Parathion
72-1909 72-1924
2,3,6-TBA
72-1954
Rat
Aldrin
72-1480
Amo 1618
72-1764
Captan
72-1513
Carbaryl
72-1496 72-1513
72-1527 72-1964
72-1993
Chlordane
72-1708
Chlormequat chloride
72-1768
Chlorpropham
72-1513
DDT
72-1471 72-1496
72-1513 72-1527
72-1533 72-1907
72-1910 72-1964
DDT isomers
72-1471
DFP
72-1441 72-1929
Diazinon
72-1513
Dichlorvos
72-1527 72-1906
Dieldrin
72-1466 72-1480
Dioxins
72-1535 72-1536
72-2003
Diquat
72-1770 72-2000
Disulfiram
72-1462
2,4-D
72-1768 72-1995
Endosulfan
72-1513
Endrin
72-1919
Ethylmethylfluorothi
72-1528
Fenitrothion
72-1527 72-1733
72-1734
Fluoroacetamide
72-1968
Hempa
72-1743
Heptachlor
72-1464 72-1737
Isopropylmethylfluor
72-1528
Lindane
72-1513 72-1919
Malathion
72-1513 72-1532
MEMC
72-1459

Mercurials
72-1464 72-1712
Methoxychlor
72-1708
Methyl bromide
72-1747
Mevinphos
72-1906
MNFA
72-1968
Monuron
72-1513
Paraquat
72-1770 72-1917
72-1918 72-2000
Parathion
72-1496 72-1964
Phosfon
72-1764
Piperonyl butoxide
72-1938
Polychlorinated biphenyls
72-1708
Sodium diethyldithiocarbama
72-1462
Soman
72-1929
Trichlorfon
72-1946
Triethylphosphate
72-1476
2,4,5-T
72-1995 72-2003
Warfarin
72-1952
Zineb
72-1946
Z-7272
72-1931

Sheep
DDT
72-1526
Di-allate
72-1922

Turkey
DDT
72-1710
Diazinon
72-1520
Ethion
72-1520
Methidathion
72-1520

Toxicity/Humans

General
72-1370 72-1596
BHC
72-1380
Captan
72-1355
Chemosterilants
72-1355
DDT
72-1355 72-1380
72-1603 72-1926
DFP
72-1441
Dieldrin
72-1380
Dioxins
72-1355
EPN
72-1450
Lindane
72-1888
Mercurials
72-1440 72-1692
MHCP
72-1450
Mirex
72-1355
Organochlorines
72-1442 72-1451
72-1681 72-1926
Organophosphates
72-1442 72-1444
72-1461 72-1681
72-1682 72-1898
72-1900
Parathion
72-1450 72-1603
72-1699
Piperonyl butoxide
72-1736
Polychlorinated biphenyls
72-1355
Thallium
72-1436

Accidental
72-1885
Dichlorvos
72-1698
Mercurials
72-1880 72-1882
72-1886
Organochlorines
72-1454 72-1705
Organophosphates
72-1453 72-1531
72-1703
Paraquat
72-1446 72-1897
Parathion
72-1454
Trichlorfon
72-1457

Intentional
Paraquat
72-1694
Thallium
72-1697 72-1884
Trichlorfon
72-1456
Tri-o-creysl phosphate
72-1697

Occupational
72-1447 72-1685
72-1687 72-1704
Aldrin
72-1878
Barban
72-1452
Benomyl
72-1889
Captafol
72-1684
Chloropicrin
72-1458
DDT
72-1636 72-1891
Di-allate
72-1452
Dichlorvos
72-1689
Dicresyl
72-1452
Dieldrin
72-1878
Dimethoate
72-1930
Endrin
72-1439 72-1683
72-1878
EPTC
72-1452
Hinosan
72-1701
Malathion
72-1445
Maneb
72-1452
Mercurials
72-1693
MNFA
72-1969
Molinate
72-1452
Organochlorines
72-1693 72-1700
Organophosphates
72-1676 72-1689
72-1691 72-1693
Parathion
72-1437 72-1675
72-1700
PCP
72-1439
Polychlorinated biphenyls
72-1700
N-Tetrachloroethylthio
72-1439

Toxicity/Wildlife

General
Dieldrin
72-1443
Mercurials
72-1605

Organochlorines
72-1605
Organophosphates
72-1605
Birds
Aldrin
72-1367
Chlordane
72-1598
DDT
72-1367 72-1438
72-1449 72-1598
72-1645 72-1658
72-1659 72-1669
DDT derived compounds
72-1438 72-1645
Dieldrin
72-1438
Endrin
72-1367
Fungicides
72-1892
Herbicides
72-1892
Mercurials
72-1438 72-1449
72-1669
Organochlorines
72-1893
Polychlorinated biphenyls
72-1449 72-1598
72-1669
TDE
72-1438 72-1645
Fish/aquatic/marine life
DDT
72-1688
Treatment of Poisoning
General
72-1447
Lindane
72-1888
Organochlorines
72-1451
Organophosphates
72-1896
Paraquat
72-1446
Parathion
72-1437
Soman
72-1953
Thallium
72-1436
Trichlorfon
72-1457
Atropine
Organochlorines
72-1883
Organophosphates
72-1453 72-1883
72-1896
Parathion
72-1699
Trichlorfon
72-1456
BAL
Mercurials
72-1886
Methyl bromide
72-1747
Chloral hydrate
Organochlorines
72-1883
Organophosphates
72-1883
Dialysis
Lead
72-1680
Mercurials
72-1680
Sodium chlorate
72-1680
Dipyroxime
Trichlorfon
72-1456
Glutathione
Methyl bromide
72-1747
Isonitrosine
Organophosphates
72-1721
N-Acetylpenicillamine
Mercurials
72-1886
Oximes
Organophosphates
72-1706
Pralidoxime
EPN
72-1450
MHCP
72-1450
Organophosphates
72-1453 72-1896
Parathion
72-1450 72-1699
Pyrodiphenium bromide
Organophosphates
72-1444
TMB-4
Organophosphates
72-1721
Toxogonin
Parathion
72-1699
2-Mercaptopropionylglycine
Organophosphates
72-1444

Abbruzzese, L.
72-2031
Abdel-Gawad, H. A.
72-1903
72-1914
Abe, Y.
72-1732
72-2021
Absalyamov, I. P.
72-1542
Abu-El-Haj, F. J.
72-2034
Adamczyk, E.
72-1772
Adams, Jr., R. S.
72-1672
Addison, R. F.
72-2023
Adkins, Jr., T. R.
72-1515
Akasu, F.
72-1380
Aleksandrova, L. G.
72-1452
Alexander, M.
72-1982
Alford, A. L.
72-1793
Almeida, J.
72-1779
Alvarez-Cervera, J.
72-1905
Amaro, R. G.
72-1779
Amos, R. A.
72-1361
Andersen, K. J.
72-1509
72-1510
Anderson, J. M.
72-1985
Anderson, J. P. E.
72-1713
Andrawes, N. R.
72-1507
Angermann, R.
72-1820
Ansari, A. N.
72-1904
Araki, S.
72-1694
72-1731
72-1788
72-1967
Arfert, H.
72-1847
Argauer, R. J.
72-1570
Arias, I. M.
72-1475
Armstrong, F. A. J.
72-1844
Armstrong, J. F.
72-1676
Asanuma, S.
72-1642
72-1643
Ashdown, D.
72-1653
Asquith, D.
72-1421
Athie, E.
72-1674
Atsumi, K.
72-1439
72-1693
Aue, W. A.
72-1810
Awasthi, M. D.
72-1388
Ayllon, R. A.
72-1668
Aziz, D.
72-1705
Azum-Gelade, M. C.
72-1535
72-1536
Baba, H.
72-1551
Bader, J.-C.
72-1970
Bagozzi, W. M.
72-1838
Baig, M. M. H.
72-1789
Bailey, C. F.
72-1653
Bailey, D. R.
72-1800
Bajgar, J.
72-1974
72-1996
Baker, H. J.
72-1743
Bakke, J. E.
72-1506
Ballee, D. L.
72-1618
Ballschmiter, K.
72-1530
72-1842
72-1913
Bareicha, I.
72-1734
Barkov, A. M.
72-1457
Barnett, B. D.
72-1853
Baron, R. L.
72-1983
Barrett, G. C.
72-1517
Bartha, R.
72-1502
Barthel, W. F.
72-1886
Bartley, W. J.
72-1981
Batterton, J. C.
72-1751
Baugh, E. R.
72-1618
Baynova, A.
72-1770
Bayon, N.
72-1591
Bazzi, B.
72-1668
72-2031
Beasley, A. G.
72-1963
Beck, J.
72-1508
Becker, H. G.
72-1820
Behrens, R.
72-1858
Beierwaltes, W. H.
72-1904
Beitz, H.
72-1382
72-1666
72-1820
72-1848
Belcher, R. S.
72-2035
Belliveau, P. E.
72-2006
Bennett, L. G. I.
72-1802
Benson, W. W.
72-1435
Benville, Jr., P. E.
72-1908
Beresi, V.
72-1540
Berger, D. D.
72-1657
Bergmann, E. D.
72-1816
Beroza, M.
72-1516
Bevenue, A.
72-1385
Beyer, N.
72-1473
Bhatia, S. C.
72-1466
Bidleman, T. F.
72-1794
Bielavsky, J.
72-1975
Bills, D. D.
72-1567
Bindra, O. S.
72-1387
Binenfeld, Z.
72-1706
Bingham, S. W.
72-1413
Bird, M.
72-1436
Biskind, M. S.
72-1885
Bitman, J.
72-1940
72-1941
Blazek, D.
72-1950
Blease, J. A.
72-1623
Bluethgen, A.
72-1588
Bobik, A.
72-1723
Bobo, C. B.
72-1691
Bodtker, D. J.
72-1749
Bogusz, M
72-1876
Boissier, J. R.
72-1729
Boiteau, H. L.
72-1704
Bollag, J. M.
72-1478
Bordas, S.
72-1459
Bordeleau, L. M.
72-1502
Bosik, J. J.
72-1753
Boskovic, B.
72-1706
Bostroem, S.-L.
72-1463
Boucher, F. R.
72-1622
Bour, H
72-1818
Boush, G. M.
72-1751
72-1962
Bovey, R. W.
72-2027
Bowen, C. E.
72-1817
Bowes, G. W.
72-1915
Bowman, M. C.
72-1426
72-1516
Boyd, E. M.
72-1513
Bozarth, G. A.
72-1415
Brady, M. N.
72-1395
Brankov, K.
72-1953
Brenner, K. S.
72-1572
Brestkin, A. P.
72-1766

Bridges, J. W.
72-1738
Briner, G. P.
72-2035
Brodeur, J.
72-1468
Brodie, B. B.
72-1938
Brown, D. R.
72-1476
Brown, N. C.
72-1736
72-1936
Brown, W. H.
72-1487
Brust, R. A.
72-1728
Bullivant, M. J.
72-1628
Bunyan, J.
72-1526
Bunyan, P. J.
72-1739
Burchill, D. J.
72-1399
Burkatskaya, Ye. N.
72-1452
Burke, J. A.
72-1558
Burke, M. D.
72-1738
Burkhard, M.
72-2037
Burse, V. W.
72-1782
Buu-Hoi, N. P.
72-1535
72-1536
Cade, T. J.
72-1658
Cahill, W. P.
72-1654
Camargo, L. A. A.
72-1733
72-1734
Campbell, R.
72-1813
Canlorbe, P.
72-1970
Carlson, A. R.
72-1986
Carpenter, C. P.
72-1993
72-1997
Carson, C. A.
72-1873
Casalis, J.
72-1664
Casida, J. E.
72-1735
72-1870
72-1947
Cassell, G. H.
72-1743
Casterline, Jr., J. L.
72-1712
Catuneanu, D.
72-1454
Cawthorne, M. A.
72-1526
Cecil, H. C.
72-1940
72-1941
Cento, D. P.
72-1838
Cerey, K.
72-1464
Cerny, E.
72-1539
Chadwick, R. W.
72-1490
Chamberlain, W. P.
72-1517
Chambers, H.
72-1719
Champ, B. R.
72-1625
Chancey, E. L.
72-1507
Chandler, J. H.
72-1616
Chang, H. S.
72-1931
Chanh, P-H.
72-1535
72-1536
Chao, T. C.
72-1900
Chasson, A. L.
72-1677
Chattopadhyay, A.
72-1802
Chau, A. S. Y.
72-1555
72-1783
Cheng, H.-M.
72-1870
Chenykayeva, Ye. Yu.
72-1760
Chichikalo, D. I.
72-1458
Childers, A. B.
72-1873
Chin, B. H.
72-1997
Chiu, Y. C.
72-1511
Choi, P. M. K.
72-1418
Chopra, N. M.
72-1386
Chopra, S. L.
72-1377
Christie, B.
72-1938
Chu, C. W.
72-1472
Chu, J. P.
72-1959
Cieleszky, V.
72-1390
Clark, P. H.
72-1518
Classen, M.
72-1929
Clausen, J.
72-1828
Clemente, A. E.
72-1581
Cliath, M. M.
72-1425
Cohn, K.
72-1514
Cole, D. W.
72-1614
Cole, M. M.
72-1518
Colmano, G.
72-1479
Comer, S. W.
72-1676
Conn, J. W.
72-1904
Coon, J. M.
72-1485
72-1603
72-1998
Corbin, H. B.
72-1874
Cordes, C. L.
72-1755
Cordes, E. H.
72-1775
Corley, C.
72-1427
Cortes-Marmolejo, F.
72-1905
Cosic, M.
72-1706
Costin, I.
72-1532
Cotrau, M.
72-1454
Coulson, J. C.
72-1626
Cox, J. L.
72-1621
Crabtree, A. N.
72-1626
Crabtree, R. J.
72-1507
Cranmer, M. F.
72-1489
Creason, J. P.
72-1615
Crofts, M.
72-2032
72-2033
Csaszar, L.
72-1689
Cserjesi, A. J.
72-1495
Csillag, R.,
72-1994
Cubit, D. A.
72-1963
Culikova, H.
72-1469
Curley, A.
72-1782
Cutaia, A. J.
72-1510
Cutkomp, L. K.
72-1488
72-1978
Czarnowski, W.
72-1636
72-1799
Czina, M.
72-1689
Dabrio, M. V.
72-1581
Dacke, C. G.
72-1757
Dacre, J. C.
72-1688
Daenens, P.
72-1795
Dahm, P. A.
72-1958
Damaskin, V. I.
72-1661
Damluji, S. F.
72-1882
Das, D. K.
72-1544
Das, M.
72-1610
72-1611
72-1612
Datta, K. K.
72-1919
Davis, G. K.
72-1964
Davison, K. L.
72-1500
72-1758
Day, Jr., E. W.
72-1580
Day, M. D.
72-1462
Deak, Z.
72-1389
Deans, I. R.
72-1626
Dedek, W.
72-1741
72-1930
Deichmann, W. B.
72-1926
72-1963
Deming, J. M.
72-1648
Demint, R. J.
72-1859

Dencker, I.
72-1832
Denmark, L. W.
72-1514
Denton, C. A.
72-1940
72-1941
Der Kinderen, P. J.
72-1460
Desaiah, D.
72-1488
72-1978
Desi, I.
72-1371
Dettbarn, W.-D.
72-1977
Deubert, K. H.
72-1640
Devine, J. M.
72-1564
72-1658
Dewan, B. S.
72-1388
72-1792
Dieter, C. T.
72-1547
Dikshith, T. S. S.
72-1919
Dillmann, I.
72-1745
Dimond, J. B.
72-1623
Dinamarca, M. L.
72-1465
Dirks, T.
72-1560
Djavaherian, M.
72-1674
Dobson, R. C.
72-1618
Docktor, M. M.
72-1872
Dokladalova, J.
72-2016
Dolan, J. W.
72-1785
Domschke, S.
72-1929
Domschke, W.
72-1929
Dorough, H. W.
72-2013
Druyan, Ye. A.
72-1807
Dubrawski, R.
72-1636
72-1637
Duby, R. T.
72-1533
Dunn, F. L.
72-2003
Durham, W. F.
72-1675
Dvorak, M.
72-1539
Dyadicheva, T. V.
72-1452
Earnest, F. D.
72-1908
Eda, S.
72-1687
Egawa, H.
72-1759
El-Bassiouni, E. A.
72-1680
El-Refai, A.
72-1724
Elder, J. H.
72-1950
Elliott, J. W.
72-1675
Elliott, M.
72-1735
Elms, K. D.
72-1625
Ember, M.
72-1689
Engdahl, B. S.
72-1786
Engelhardt, G.
72-1923
Engst, R.
72-1948
Enhoerning, G.
72-1917
Ennis, Jr., W. B.
72-1368
Epifanio, C. E.
72-1754
Epstein, S. S.
72-1355
Ercegovich, C. D.
72-1421
72-1422
72-1571
Erdos, G.
72-1371
Eskenazi, M.
72-1718
Estesen, B. J.
72-1654
Eto, M.
72-1920
Eue, L.
72-1748
Ezell, Jr., W. B.
72-1515
Fabacher, D. L.
72-1719
Faber, R. A.
72-1438
Fahey, J. E.
72-1618
Falk, H. L.
72-1481
Farmer, W. J.
72-1856
Faust, S. D.
72-1843
Feaster, J. P.
72-1964
Felbeck, Jr., G. T.
72-1854
Feldmann, R. J.
72-1935
Feltner, K. C.
72-1857
Findlay, E.
72-1851
Finger, E.
72-2012
Finklea, J. F.
72-1615
72-1691
72-1891
Fishbein, L.
72-1505
72-1714
72-2017
Fisher, N. S.
72-1483
Fitzpatrick, J. T.
72-1981
Fletcher, W. W.
72-1367
Florant, G. L.
72-1657
Floru, S.
72-1431
Flotard, R. D.
72-1871
Foehrenbach, J.
72-1394
72-1645
Forey, A.
72-1752
Forrest, T. J.
72-1912
Foster, G. G.
72-1825
Frank, P. A.
72-1859
Franklin, M. P.
72-1742
Freal, J. J.
72-1490
Freed, V. H.
72-1620
72-1864
Frehse, H.
72-1798
Frei, R. W.
72-1573
72-1794
72-2006
French, M. C.
72-1911
Friedman, M. A.
72-1934
Friend, M.
72-1493
Fries, G. F.
72-1420
72-1940
72-1941
Frost, K. R.
72-1654
Fryer, J. D.
72-1865
Fuhremann, T. W.
72-1867
72-1951
Fujimoto, Y.
72-1411
72-1412
72-1548
Fujita, M.
72-1846
72-2009
Fujiwara, K.
72-1639
Funazaki, Z.
72-1678
Funderburk, Jr., H. H.
72-1415
Gabica, J.
72-1435
Gadsden, R. H.
72-1891
Galin, M. A.
72-1514
Galley, D. J.
72-1849
Galluzzi, G.
72-1668
72-2031
Gardiner, A. J. S.
72-1446
Garty, O. M.
72-2011
Gasper, G. M.
72-1934
Gates, C. E.
72-1922
Gatmaitan, Z.
72-1475
Gavryuk, A. P.
72-1458
Gawhary, A. S.
72-1976
Gehrke, C. W.
72-1810
Geike, F.
72-1575
Geisman, J. R.
72-1862
Geleriu, R.
72-1532
Gesser, H.
72-1781
Getchell, A. S.
72-1623
Getzendaner, M. E.
72-1874

Ghelberg, N. W.
72-1532
Ghezzo, F.
72-1737
Ghimicescu, C.
72-1454
Giang, P. A.
72-1778
Gibson, J. R.
72-1484
Gill, R.
72-1825
Gillette, R.
72-1826
Giri, S. N.
72-1979
Glotfelty, D. E.
72-1777
Glukhovets, B. I.
72-1457
Goerlitz, D. F.
72-1561
Goerttler, K.
72-1534
Golovan', D. I.
72-1954
Gomaa, H. M.
72-1843
Gooding, R. H.
72-1780
Goodship, G.
72-1396
Goodwin, F. S.
72-1779
Gordon, C. H.
72-1420
Gordts, L.
72-1563
Gorshkov, A. I.
72-1768
Gostick, K. G.
72-1396
Goto, S.
72-1400
Goursaud, J.
72-1664
Granhall, I.
72-1597
Granick, S.
72-1774
Grant, D. L.
72-1708
72-1711
Green, J
72-1526
Green, R. E.
72-1855
Greene, E. J.
72-1994
Greenhalgh, R.
72-2016
Greichus, Y. A.
72-1492
Greve, P. A.
72-1393
Grifantini, M.
72-1477
Grim, M. E.
72-1782
Gross, W. B.
72-1479
Grossklaus, D.
72-1619
Grover, R.
72-2002
Gruebner, P.
72-1665
72-2019
Gulati, K. C.
72-1792
Guseynov, M. M.
72-1543
Gutenmann, W. H.
72-1392
72-1562
Guth, J. A.
72-2025
Guthrie, F. E.
72-1677
Guttel, C.
72-1674
Haag, D.
72-1534
Habeck, D. H.
72-1651
Hagiwara, A.
72-1642
Haider, K.
72-1397
Haile, D. G.
72-1374
Halacka, K.
72-1539
Hall, J. M.
72-1462
Hall, R. C.
72-1785
Hammer, D. I.
72-1615
Hance, R. J.
72-1399
Handa, S. K.
72-1388
Hanks, A. R.
72-1786
Hannan, E. J.
72-1567
Haque, R.
72-1620
Hargrove, R. S.
72-1414
Haring, R. C.
72-1833
Harris, C. R.
72-1649
72-1650
Harris, L. S.
72-1514
Harris, R. J.
72-2032
72-2033
Harris, S. J.
72-1941
Harris, W. G.
72-1784
Harrison, D. L.
72-1895
Hartisch, J.
72-1848
Haselein, I.
72-1666
72-1821
Hashimoto, Y.
72-1892
72-1893
72-1894
Hassall, K. A.
72-1912
Hastings, C. R.
72-1810
Hatfield, C. T.
72-1985
Hattula, M. L.
72-1796
Haufe, W. O.
72-2016
Haufler, M.
72-1922
Hauser, T.
72-1615
Haussdoerfer, M.
72-1666
Hayamizu, K.
72-1583
Hays, H.
72-1449
Hazard, R.
72-1729
Heeschen, W.
72-1588
Hegyi, E.
72-1702
Heinisch, E.
72-1666
72-1667
72-1848
Helling, C. S.
72-1547
Hellwig, A.
72-2007
Hempel, D.
72-2007
Henderson, P. T.
72-1746
Herfs, W.
72-1604
Herigstad, R. R.
72-1473
Hernandez, E.
72-1863
Herrett, R. A.
72-1507
Herrick, R. B.
72-1508
72-1727
72-1944
Herzel, F.
72-1670
Hesselberg, R. J.
72-1556
Heuser, S. G.
72-1396
Hiltibran, R. C.
72-1916
Himel, C.
72-1560
Hines, N. W.
72-1814
Hinman, A. R.
72-1886
Hinners, T.
72-1615
Hirakawa, M.
72-1897
Hiraki, K.
72-1450
Hirsch, I.
72-1354
Hirwe, A. S.
72-1980
Hitchcock, B. E.
72-1383
Hodgson, E.
72-1470
Hodgson, G. C.
72-1728
Holder, G. M.
72-1723
72-2004
Hollaender, A.
72-1972
Holland, P.
72-1925
Holmes, D. C.
72-1574
Hontani, I.
72-1829
Hood, R. D.
72-1494
Hopkins, D. E.
72-1517
Hopkins, T. L.
72-1724
Horowitz, M.
72-2026
Horvath, A.
72-1389
Horvath, R. S.
72-1617
Hrdina, P. D.
72-1471
72-1907
72-1910

Huang, J. C.
72-1601
Hudakova, G.
72-1702
Huddleston, E. W.
72-1653
Hulin, M.
72-2026
Humiczewska, M.
72-1973
Hunn, J. B.
72-1945
Hunt, L. M.
72-1922
Hunter, J.
72-1878
Hussain, A.
72-1789
72-1790
Huston, B. L.
72-1569
Hutzinger, O.
72-1418
72-2008
Hyland, J. R.
72-1838
Hylin, J. W.
72-1385
Ide, T.
72-1643
Igue, K.
72-1856
Iijima, T.
72-1631
Ikeda, S.
72-1750
Ikramov, L. T.
72-1582
72-1805
Iliff, M.
72-1824
Illes, S.
72-1776
Imaizumi, K.
72-1439
72-1693
Ishiguro, T.
72-1960
Ishii, Y.
72-1411
72-1550
Ishikawa, S.
72-1703
Ishikawa, T.
72-1898
Istrate, E.
72-1454
Ito, R.
72-1931
Ivanova, Z. V.
72-1452
Ivanova, Zh. M.
72-1528
Ivemark, B. I.
72-1917
72-1918
Iverson, F.
72-1524
Iwabuchi, T.
72-1694
Iwan, J.
72-1984
Iwasaki, I.
72-1450
Iwashima, K.
72-1846
Izumiyama, T.
72-1686
Jackson, M. D.
72-1677
Jackson, W. B.
72-1952
Jacobsen, A. M.
72-1872
Jagnow, G.
72-1397
Jain, M. K.
72-1775
Jamieson, W. D.
72-1418
Janes, N. F.
72-1709
72-1735
Jaworski, J.
72-1806
Jayaram, M.
72-1545
Jefferies, D. J.
72-1845
72-1911
Jennings, R. W.
72-1782
Jenny, E.
72-1455
Jervis, R. E.
72-1802
Jirik, V.
72-1469
Job, J.-C.
72-1970
Johansson, R. G.
72-1463
Johnson, E. L.
72-1495
Johnson, J. L.
72-1556
Johnson, Jr., J. C.
72-1516
Johnson, M. K.
72-1525
Jones, D. W.
72-1497
Jung, W.
72-1847
Juszkiewicz, T.
72-1875
Kaai, M.
72-1641
72-1747
Kacew, S.
72-1910
Kado, M.
72-1381
Kadoum, A. M.
72-1652
Kagawa, R.
72-1430
Kakihara, Y.
72-1788
72-2020
Kakimoto, S.
72-1444
Kamada, T.
72-1835
Kamata, T.
72-1773
72-1897
Kanagasuntheram, R.
72-1932
Kanazawa, J.
72-1630
Kanda, M.
72-1731
72-1732
72-1788
72-2020
72-2021
Kanemitsu, J.
72-1444
Kapoor, I. P.
72-1980
Karkos, J.
72-1884
Karlog, O.
72-1796
Kataoka, S.
72-1731
72-1788
72-2020
Kato, M.
72-1740
Katz, A.
72-1609
Kaufman, D. D.
72-1547
72-1984
Kaufman, W. M.
72-1567
Kaukeinen, D.
72-1952
Kaul, R.
72-1715
Kawada, S.
72-1381
Kawahara, T.
72-1401
72-1402
72-1403
72-1404
72-1405
72-1406
72-1407
72-1408
72-1409
72-1410
72-1411
72-1548
72-1549
72-1552
72-1553
Kawakita, H.
72-1987
72-1988
72-1989
Kawamura, H.
72-1931
Kawanishi, A.
72-1642
72-1643
Kawasaki, T.
72-1731
72-2021
Kawatski, J. A.
72-1726
72-1934
Kaye, S.
72-1881
Kearney, P. C.
72-1423
Kearns, C. W.
72-1499
Keil, J. E.
72-1696
72-1891
Keith, L. H.
72-1793
Kelley, T. W.
72-1385
Kelly, W. A.
72-1496
Kenney, A. D.
72-1757
Kerr, J. D.
72-1625
Kevorkian, J.
72-1838
Khalifa, S.
72-1568
Khan, M. S.
72-1789
72-1790
Khan, S. U.
72-1391
Khera, K. S.
72-1995
Khramova, E. A.
72-1721
Khullar, P. C.
72-1377
Kielczewski, W.
72-1803
Kimmel, E. C.
72-1735

Kingsbury, P. J.
72-1749
Kintanar, Q. L.
72-1584
Kirchner, K.
72-1666
Kirkland, K.
72-1865
Kirkwood, B. C.
72-2001
Kirsch, E. J.
72-1959
Kishikawa, J.
72-1444
Kissam, J. B.
72-1853
Kita, G.
72-1732
72-2021
Klein, W.
72-1671
72-1715
72-1716
Klingebiel, U. I.
72-1984
Klosterman, H. J.
72-1655
Klotz, H. P.
72-1763
Knaus, J. P.
72-1364
Knox, F. E.
72-1516
Koch, R. E.
72-1488
72-1978
Koeman, J. H.
72-1443
Koivusaari, J.
72-1669
Kojima, K.
72-1836
Kolbezen, M. J.
72-2034
Komissarenko, V. P.
72-1767
Kondoh, H.
72-1687
Kondratyuk, V. I.
72-1528
Konuma, S.
72-1405
Koons, J. R.
72-1580
Korte, F.
72-1671
72-1716
Kosaka, S.
72-1921
Kosarev, V. A.
72-1456
Kramer, J. P.
72-1437
Krampl, V.
72-1480
Krasnicki, K.
72-1806
Krasyuk, N. N.
72-1769
Kratky, B. A.
72-1546
Krechniak, J.
72-1636
72-1637
Kreybig, T. von
72-1818
Krieger, H. G.
72-1677
Krijnen, C. J.
72-1513
Krishna, G.
72-1938
Kroger, M.
72-1378
Kruglova, E. E.
72-1760
Kudo, A.
72-1834
Kudo, N.
72-1682
Kudzina, G. D.
72-1954
Kuliyeva, A. M.
72-1765
72-1957
Kunz, S. E.
72-1520
Kuratsune, M.
72-1430
Kureha, Y.
72-1405
Kuriyama, T.
72-1834
Kurstak, E.
72-1585
Kurtz, A. P.
72-1981
Kushida, K.
72-1583
Kusuki, T.
72-1444
Laamanen, A.
72-1669
Lahue, D. W.
72-1652
Landauer, W.
72-1720
Landmann, W. A.
72-1873
Lapchenko, V. S.
72-1434
Larini, L.
72-1733
72-1734
Larson, J. D.
72-1506
Law, L. M.
72-1561
Lawrence, J. P.
72-1573
Le Grand, H. E.
72-1860
Ledbetter, J. O.
72-1887
Leduc, R.
72-1852
Lee, G. P.
72-1622
Leefe, J. S.
72-1861
Lefstad, S.
72-1879
Legator, M. S.
72-1815
Lehotzky, K.
72-1459
Leib, E.
72-1600
Leighty, E. G.
72-1509
Leiper, J. W. G.
72-1526
Lembcke, G.
72-1666
72-1821
Lembi, C. A.
72-1950
Leuck, D. B.
72-1426
72-1516
Levenbook, L.
72-1465
Levi, A. J.
72-1475
Levi, I.
72-1557
72-1559
Lewallen, M. J.
72-1662
Li, G.-C.
72-1854
Liang, T. T.
72-1647
Lichtenstein, E. P.
72-1647
72-1713
72-1867
72-1951
Lieberman, L. M.
72-1904
Liebmann, R.
72-2007
Likosky, W. H.
72-1886
Lillie, R. J.
72-1940
72-1941
Lincer, J. L.
72-1658
Linder, R. E.
72-1782
Lindsey, J. R.
72-1743
Ling, G. M.
72-1471
72-1910
Linyuchev, M. N.
72-1721
Lipkan, G. N.
72-1528
Lisella, F. S.
72-1602
Lisk, D. J.
72-1392
72-1562
72-1656
Lisowe, R. W.
72-2003
Liu, S. Y.
72-1478
Lloyd-Jones, C. P.
72-1357
Locke, B. K.
72-1983
Lockman, D. C.
72-1943
Long, K. R.
72-1602
Longbrake, T. D.
72-1663
Lorber, M.
72-1933
Lord, K. A.
72-1789
72-1790
Lubberink, A. A. M. E.
72-1460
Lubkowitz, J. A.
72-1787
Ludke, J. L.
72-1484
Ludvik, G. F.
72-1648
Luquet, F. M.
72-1664
Lusk, C. I.
72-1484
Luzhnikov, Ye. A.
72-1456
Lyle, C. D.
72-1883
Lynch, W. T.
72-1485
Machin, A. F.
72-1709
MacDonald, W. E.
72-1963
MacLennan, K. J. R.
72-1443
Maddox, J.
72-1822
Maeda, H.
72-1410

Maeda, T.
72-1635
Maes, R.
72-2014
Magos, L.
72-1797
Mahmood, G.
72-1645
Maibach, H. I.
72-1935
Major, M.
72-1371
Majumder, S. K.
72-1545
Maliszewski, M. J.
72-1522
Malmqvist, E.
72-1917
Marchand, C.
72-1468
Marchuk, R. Ya.
72-1528
Markkula, M.
72-1363
Markle, J. C.
72-1554
Marks, R. H. L.
72-1775
Marrow, Jr., G. S.
72-1420
Marstrand, P. K.
72-1365
Martelli, S.
72-1477
Martin, G. C.
72-1903
72-1914
Martin, J. P.
72-1856
Martinez Reyes, C.
72-1596
Martinez, M.
72-1863
Martinez, P. M.
72-1863
Masuda, Y.
72-1430
Masuko, M.
72-1759
Mata, E.
72-1998
Mathew, T. V.
72-1544
Mathur, D. S.
72-1486
Matlib, M. A.
72-2001
Matokhnyuk, L. A.
72-1452
Matschiner, J. T.
72-1744
Matsu-ura, K.
72-1407
Matsu-ura, S.
72-1931
Matsumoto, K.
72-1788
Matsumoto, M.
72-1639
Matsumura, F.
72-1751
72-1962
Matsunaga, A. K.
72-1779
Matsunaga, H.
72-1890
Matsunaga, K.
72-2023
72-2024
Matsushima, S.
72-1642
72-1643
Mattheis, I.
72-1847
Matthews, H. B.
72-1505
Matthey, E.
72-1379
Mattocks, A. M.
72-1680
Matyushina, V. I.
72-1452
Maxwell, J. D.
72-1878
McAleese, D. M.
72-1360
McBain, J. B.
72-1947
McCully, K. A.
72-1579
McElligott, T. F.
72-1992
McGuire, R. R.
72-1871
McIntyre, A. R.
72-1440
McKinley, W. P.
72-1995
McKinney, J. D.
72-1505
72-1714
McKone, C. E.
72-1399
McLeod, H. A.
72-1566
Medved', I. L.
72-1452
Menandez, M. I.
72-1581
Mendoza Hernandez, P.
72-1596
Menges, R. M.
72-1663
Merkle, M. G.
72-1414
72-2027
Mestechkina, A. Ya.
72-1722
Mestres, R.
72-1776
Metcalf, R. L.
72-1499
72-1980
Michaux, P.
72-1704
Mickelsen, O.
72-1473
Minagawa, K.
72-1837
Mindszenty, L.
72-1689
Mirna, A.
72-1646
Misra, S. S.
72-1388
Mita, K.
72-1693
Mitchell, J. R.
72-1938
Mitra, S. N.
72-1544
Mittag, T. W.
72-1514
Miyachi, Y.
72-2030
Miyamato, Y.
72-1565
Miyamoto, J.
72-1461
72-1961
Miyashita, H.
72-1693
Miyazaki, S.
72-1728
Mizutani, T.
72-1639
Mobbs, R. F.
72-1885
Modee, J.
72-1917
72-1918
Moergeli, E.
72-1841
Moffett, G. E.
72-1501
Moku, M.
72-1403
72-1553
Molnar, J.
72-1906
Montgomery, M.
72-1864
Mori, S.
72-1444
Morita, K.
72-2023
72-2024
Morita, R.
72-1904
Morre, D. J.
72-1950
Moskowitz, J.
72-1938
Mosser, J. L.
72-1483
Motas, C.
72-1605
Mozai, T.
72-1442
Mueller, K.
72-1572
Mukherjee, A. K.
72-1544
Mull, R. L.
72-1979
Muller, H. D.
72-1943
Mumma, R. O.
72-1568
Munson, T. O.
72-1419
Muramatsu, T.
72-1687
Murano, A.
72-1801
Murata, T.
72-1384
Muro, T.
72-1442
Murphy, S. D.
72-1476
Murrell, E. A.
72-1526
Musacchia, X. J.
72-1757
Na'isa, B. K.
72-1443
Nagami, H.
72-1683
Nakagawa, A.
72-1897
Nakamura, H.
72-1401
72-1402
72-1403
72-1404
72-1405
72-1406
72-1407
72-1411
72-1412
72-1551
Nakamura, K.
72-1687
Nakamura, R.
72-1444
Nakanishi, M.
72-1834
Narimoto, T.
72-2023
72-2024
Nash, R. G.
72-1784

Naumann, K.
72-1761
72-1927
Neal, R. A.
72-1909
72-1924
72-1999
Neilson, J. T.
72-1710
Nelson, D. C.
72-1655
Nesti, V.
72-1668
72-2031
Neubert, D.
72-1745
Neururer, H.
72-1632
72-1633
Nicholson, H. P.
72-1811
Nickey, L. N.
72-1886
Nikolayev, A. I.
72-1771
Nishimura, H.
72-1679
72-1971
Nishiyama, H.
72-1904
Noguchi, T.
72-1921
Nogueira, D. P.
72-1674
Novikov, Yu. V.
72-1946
Nowicki, T. W.
72-1557
72-1559
Nowlan, E.
72-1794
Nozue, I.
72-1687
Nuuja, I.
72-1669
O'Brien, R. D.
72-1511
Oarda, M.
72-1699
Oba, S.
72-1801
Obst, A.
72-1819
Ogata, S.
72-1730
Ogata, Y.
72-2020
Ogino, Y.
72-2023
72-2024
Ohba, Y.
72-1687
Ohishi, S.
72-1695
Ohnishi, M.
72-1565
Oho, S.
72-2021
Okada, H.
72-1897
Okada, M.
72-1683
Okey, R. W.
72-1833
Oki, K.
72-1965
Okuda, I.
72-1381
Okun'kov, P. S.
72-1372
Onyshchenko, D. S.
72-1767
Oshiba, K.
72-1987
72-1988
72-1989
Oto, K.
72-1453
Otsuka, J.
72-1531
Ottolenghi, A.
72-2000
Overcash, R. G.
72-1743
Owen, D. A. A.
72-1462
Owens, J. C.
72-1653
Paleg, L.
72-1764
Palmer, J. S.
72-1474
72-1519
72-1922
Palokangas, R.
72-1669
Pank, F.
72-1382
Papworth, D. S.
72-1592
Parker, W. C.
72-1787
Parkes, D.
72-1925
Parkhomenko, T. V.
72-1766
Parr, J. F.
72-1868
Pasi, A.
72-1455
Pastia, A.
72-1454
Pathak, M. D.
72-1504
Patil, K. C.
72-1962
Patocka, J.
72-1975
Pattenden, G.
72-1628
Patterson, J. D. E.
72-2001
Patyk, S.
72-1762
Paulian, F.
72-1431
Paulson, G.
72-1436
Paulson, G. D.
72-1872
Pelikan, Z.
72-1737
Persson, K.
72-1832
Persson, T.
72-1467
Pestova, A. G.
72-1452
Peters. D. A. V.
72-1471
Peters, D. C.
72-1812
Pettigrew, P. J.
72-1991
Pfaender, F. K.
72-1982
Philip, H. G.
72-1780
Phillips, F. T.
72-1629
Phillips, W. E. J.
72-1708
72-1711
Phillips, W. M.
72-1857
Philpot, R. M.
72-1470
Piedade, J. P.
72-1564
72-1779
Piedade, M. P.
72-1564
72-1779
Pierce, P. E.
72-1886
Pietsch, R. L.
72-1691
Pirie, A.
72-1717
Pizzi, T.
72-1540
Plakhova, I. G.
72-1966
Plato, C.
72-2015
Plimmer, J. R.
72-1423
72-1984
Pogacnick, J.
72-1668
Pokorny, J.
72-1469
Poland, J. J.
72-1853
Polizu, A.
72-1431
Poloz, D. D.
72-1372
Pongracz, M. P.
72-1906
Poonacha, K. B.
72-1500
Pope, G. G.
72-1937
Popov, L. G.
72-1458
Posner, H. S.
72-2000
Potts, G. R.
72-1626
Powell, D. F.
72-1396
Pozo, H. del.
72-1540
Prahlad, K. V.
72-1942
Pratt, H. M.
72-1438
Preiss, D.
72-1534
Preussmann, R.
72-1698
Price, C. E.
72-1506
Priester, L. E.
72-1615
Prokopchuk, V. O.
72-1767
Prospero, J. M.
72-1357
Prout, T.
72-1825
Ptashne, K. A.
72-1909
Qadri, S. S. H.
72-1791
Quick, M. P.
72-1709
Quraishi, M. S.
72-1655
Radeleff, R. D.
72-1520
Radomanski, T.
72-1875
Ragab, M. T. H.
72-1861
Raghavan, N. G. S.
72-1610
72-1611
72-1612
Rakin, D.
72-1706
Rammell, C. G.
72-1627
Randolph, N. M.
72-1433

Reed, J. K.
72-1853
Rees, J. S.
72-1521
Reid, W. D.
72-1938
Reinert, F. E.
72-1839
Reinke, J.
72-1781
Rengei, B.
72-1689
Renier-Cornec, A.
72-1729
Renier, E.
72-1729
Reyes, H.
72-1475
Reynoso Zuniga, E.
72-1596
Reznikov, A. G.
72-1538
Richardson, A.
72-1878
Richert, E. P.
72-1942
Richey, Jr., F. A.
72-1981
Richter, R.
72-1879
Rickett, F. E.
72-1936
Rijksen, H. D.
72-1443
Rijnberk, A.
72-146C
Rio, A. del
72-1540
Risebrough, R. W.
72-1438
72-1449
72-1613
72-1657
Ritter, W. F.
72-1375
Rivas Llamas, R.
72-1596
Rivera, G.
72-1540
Roberts, D. V.
72-1441
Robertson, B.
72-1917
72-1918
Robinson, D. W.
72-1366
Robinson, J.
72-1626
72-1878
Robinson, J. R.
72-1624
Rodgers, C. A.
72-1955
Rogers, H.
72-1709
Rosen, J. D.
72-1502
Rosival, L.
72-1464
Roth, J. A.
72-1924
Routh, J. D.
72-1417
Rowe-Rowe, D. T.
72-1482
Rowe, V. K.
72-2003
Rozengart, V. I.
72-1765
72-1957
Rudge, A. J. B.
72-1592
Rueckert, W.
72-1530
Russo, F.
72-1763
Ruttkayova, J.
72-1464
Ryan, A. J.
72-1723
72-2004
Saad, W. A.
72-1733
72-1734
Sabine, J. R.
72-1764
Sacher, R. M.
72-1648
72-1928
Sadriyeva, R. V.
72-1542
Safe, S.
72-2008
Saha, J. G.
72-1428
Saint-Ruf, G.
72-1535
72-1536
Saito, T.
72-1960
Sajko, B.
72-1842
Sakagami, Y.
72-2009
Sakata, M.
72-1920
Sakurai, H.
72-1551
Salam, M.
72-1720
Sanders, H. O.
72-1616
Sandi, E.
72-1852
Sandifer, S. H.
72-1696
72-1891
Sans, W. W.
72-1649
72-1650
Santa Maria, H. C.
72-1668
Sare, W. M.
72-1445
Sarli, C. A.
72-1668
Sasaki, T.
72-1931
Sasayama, T.
72-1920
Sasinovich, L. M.
72-1527
Sassa, S.
72-1774
Satoh, S.
72-1583
Satrapinsky, Yu. F.
72-1721
Sattel, P.
72-1572
Sauer, H. H.
72-1503
Sauter, E. A.
72-1939
Savitt, L. E.
72-1889
Scanlon, J.
72-1692
Schafer, E. W.
72-1990
Scheid, W.
72-1697
Schiefner, K.
72-1389
Schlinke, J. C.
72-1519
72-1922
Schmedding, D.
72-1620
Schmidt, F.
72-1741
Schmulbach, J. C.
72-1726
Schuetz, R. D.
72-1871
Schulz, K. R.
72-1867
Schuphan, I.
72-1842
72-1913
Scifres, C. J.
72-2027
Scott, D.
72-1688
Scott, H. G.
72-1602
Scripcaru, G.
72-1454
Seagran, H. L.
72-1839
Seba, D. P.
72-1357
Sedov, A. I.
72-1541
Seefeld, F.
72-1382
72-1666
72-1848
Segawa, M.
72-1703
Seiber, J. N.
72-1554
Seki, R.
72-1683
Sekida, H.
72-2022
Sell, J. L.
72-1500
72-1758
Seniow, A.
72-1762
Sergeant, M.
72-1950
Sesque, G.
72-1535
72-1536
Sethunathan, N.
72-1504
Severson, K. E.
72-1492
Shakespeare, J.
72-1925
Sharma, S. C.
72-1466
Sharp, C. W.
72-2000
Shaver, R.
72-1413
Shaw, S. R.
72-1673
Sherman, L. R.
72-1386
Sherman, M.
72-1508
72-1727
72-1944
Shibuya, M.
72-1576
Shikata, H.
72-1759
Shimada, Y.
72-1442
Shimano, N.
72-1565
Shmeleva, V. G.
72-1765
72-1957
Shoyama, M.
72-2030
Shtarkas, Ye. M.
72-1638
Sieczka, J. B.
72-1656

Silva, L. P.
72-1779
Simmonds, P. J.
72-1369
Simpson, C. F.
72-1710
Sinclair, D.
72-1599
Singh, N. N.
72-1610
72-1611
Singha, P.
72-1612
Singhal, R. L.
72-1471
72-1907
72-1910
Sinnappa, S.
72-1808
Siyali, D. S.
72-1395
Skrentny, R. F.
72-2013
Skromme-Kadlubik, G.
72-1905
Slama, K.
72-1356
Sleight, S. D.
72-1902
Smies, M.
72-1443
Smith, A. E.
72-1398
72-1866
Smith, D. C.
72-1852
Smith, F.
72-1427
Smith, F. D.
72-1677
Smith, J. M.
72-1949
Smith, Jr., W. M.
72-1887
Smith, R. H.
72-1655
Smith, S.
72-1868
Smyth, Jr., H. F.
72-1993
Snyder, R. D.
72-1880
Sobotka, T.
72-2005
Soeda, Y.
72-1921
Soedergren, A.
72-1429
Soga, T.
72-2023
72-2024
Sohda, T.
72-1687
Soller, B.
72-1831
Solly, S. R. B.
72-1896
Soos, K.
72-1390
Sopher, D.
72-1720
Souza Lima, A. A.
72-1779
Souza, D. de A.
72-1779
Sparschu, G. L.
72-2003
Spencer, W. F.
72-1425
72-1856
Springfield, A. C.
72-1498
Sram, R. J.
72-1370
St. John, Jr., L. E.
72-1562
Staiff, D. C.
72-1676
Stalling, D. L.
72-1955
Stancliffe, T. C.
72-1717
Stanosz, S.
72-1973
Stec, J.
72-1875
Steele, E. E.
72-1939
Steen, K. B.
72-1571
Stein, M. L.
72-1477
Stepanchenko, N. G.
72-1452
Stevens, J. T.
72-1361
Stewart, D.
72-1839
Stewart, D. A.
72-1878
Stickley, B. D. A.
72-1383
Stolzenberg, R. L.
72-1707
Straus, H.
72-1532
Strayer, J. R.
72-1651
Stull, J. W.
72-1487
Su, G. C. C.
72-1424
Sugaya, T.
72-1451
72-1644
72-1685
Sullivan, D.
72-1645
Sullivan, L. J.
72-1997
Suzuki, S.
72-1537
Swann, C. W.
72-1858
Sydymanov, B. B.
72-1577
Szeberenyi, J.
72-1906
Sztankayne Gulyas, M.
72-1371
Takagi, S.
72-1899
72-1968
72-1969
Takahama, K.
72-1732
72-1788
72-2020
72-2021
Takahashi, I.
72-1830
Takahashi, M. T.
72-1509
Takahashi, S.
72-1384
Takamatsu, C.
72-2022
Takami, F.
72-1635
Takano, T.
72-1444
Takano, Y.
72-1681
Takeda, A.
72-2022
Takeo, T.
72-1576
Takeshita, R.
72-1804
Talanov, G. A.
72-1372
Tamez, S.
72-1663
Tanabe, H.
72-1376
72-2022
72-2028
Tanimura, T.
72-1971
Tanji, J.
72-1740
Tappan, W. B.
72-1677
Taranova, N. P.
72-1760
Tarjan, R.
72-1390
Tashpulatov, A. Yu.
72-1582
72-1805
Tawfik, M. S.
72-1780
Taylor, A.
72-1739
Taylor, D. A.
72-1361
Taylor, K.
72-1441
Teetes, G. L.
72-1433
Teli, P. M
72-1810
Temple, S. A.
72-1659
Terol, J. P.
72-1863
Thakre, S. K.
72-1792
Thibault, E.
72-1763
Thijssen, J. H. H.
72-1460
Thill, R. E.
72-1492
Thimm, H. F.
72-2035
Thomas, J. A.
72-1361
Thompson, C. J.
72-1627
Thompson, J. F.
72-1886
Thompson, Jr., L.
72-1956
Thompson, N. P.
72-1710
Thuan, C. E.
72-1808
Tibaldi, E.
72-1373
Tietz, H.
72-1748
Tikriti, S.
72-1882
Tildyova, K.
72-1464
Timmons, F. I.
72-1859
Tjaij, J. K.
72-1705
Tkachuk, R.
72-1634
Todd, T. M.
72-1785
Tohdoh, M.
72-1687
Toida, S.
72-1931
Tolle, A.
72-1588
Tolot, F.
72-1704
Tomatis, I.
72-1674

Tomkowiak, J.
72-1803
Tong, S. C.
72-1562
Torii, I.
72-2023
72-2024
Tosa, Y.
72-1683
Toshimitsu, T.
72-1732
Townsend, M. G.
72-1739
Toyoda, I.
72-1960
Trainer, D. O.
72-1493
Trebicka-Kwiatkowska, B.
72-1875
Triolo, A. J.
72-1998
Tripathi, R. K.
72-1511
Trommer, R.
72-2018
Tron'ko, M. D.
72-1529
Trudgill, P. W.
72-1521
72-1522
72-1523
Tsuda, T.
72-1444
Tsunenari, S.
72-2020
Turchyn, I. S.
72-1767
Turk, A.
72-1833
Turnell, D. C.
72-1523
Tyszkiewicz, K.
72-1936
Uchida, A.
72-1687
Uchiyama, J.
72-1687
Uchman, W.
72-1803
Udeaan, A. S.
72-1387
Ueda, K.
72-1747
Uete, T.
72-1565
Ueyama, A.
72-1759
Uk, S.
72-1560
Umeda, I.
72-1801
Unno, I.
72-1687
Uono, K.
72-1703
Ushio, K.
72-1694
Usmanova, I. Ya.
72-1771
Uthe, J. F.
72-1781
Uzoukwu, M.
72-1902
V''lcheva, V.
72-1770
Vaisman, S.
72-1540
Valdes, E.
72-1465
Vallotton, W. W.
72-1691
van Halteren, P.
72-1725
van Hollebeke, E.
72-1838
Van Middelem, C. H.
72-1651
72-1964
Van Tienhoven, A.
72-1533
Varga, A.
72-1906
Vargova, M.
72-1464
Vaughn, W. K.
72-1999
Vega, O. D.
72-1668
Velle, W.
72-1598
Venkitasubramanian, T. A.
72-1466
Vergara, G.
72-1436
Verma, S.
72-1388
Vermeer, K.
72-1844
Versino, B.
72-1589
Vihko, V.
72-1669
Vij, S.
72-1932
Villeneuve, D. C.
72-1708
Virtamo, M.
72-1700
Visweswariah, K.
72-1545
Vodenicharova, M.
72-1718
Volkert, W. A.
72-1757
von Rosen, G.
72-1832
Vorob'yeva, N. M.
72-1434
Voronina, L. Ya.
72-1527
Voss, G.
72-2025
72-2037
Voytenko, G. A.
72-1452
Wada, T.
72-1405
Wagenbreth, D.
72-2018
Wagstaff, D. J.
72-1757
Wakahara, S.
72-1635
Wakai, S.
72-1837
Wakatsuki, S.
72-1643
Wales, P. J.
72-1566
Walker, A.
72-1850
Wallnoefer, P.
72-1923
Wangenheim, M.
72-1455
Ward, C. R.
72-1653
Ward, P.
72-1937
Ware, G. W.
72-1654
72-1877
Warren, G. F.
72-1546
Wassermann, D.
72-1674
Wassermann, M.
72-1674
Watanabe, S.
72-1684
Watanabe, T.
72-1400
72-1411
Watkins, J. H.
72-1526
Watson, M.
72-1435
Watson, T. R.
72-2004
Wattal, B. L.
72-1610
72-1611
72-1612
Watts, B. B.
72-1595
Weaver, R. J.
72-1903
72-1914
Webb, R. F.
72-1570
Weber, K.
72-1578
Weiden, M. H. J.
72-1507
Weil, C. S.
72-1993
Weinmann, W.
72-2012
Weisgerber, I.
72-1715
72-1716
Welsch, F.
72-1977
Wenzel, K.-D.
72-1930
Whitehair, C. K.
72-1473
Whitehead, C. C.
72-1991
Whiteoak, R. J.
72-2032
72-2033
Whiting, F. M.
72-1487
Whitley, J. R.
72-1813
Whitten, M. J.
72-1825
Wickstroem, G.
72-1606
Widdus, R.
72-1521
72-1522
72-1523
Wiebe, L. I.
72-2004
Wildish, D. J.
72-1491
Wilkinson, R. J.
72-1555
Williams, C. H.
72-1712
Williams, D. A.
72-1878
Williams, I. H.
72-2036
Williams, P. P.
72-1707
Willis, D. E.
72-2029
Wilson, A.
72-1441
Wit, S. L.
72-1393
Witkonton, S.
72-1421
72-1422
72-1571
Wohlgemuth, R.
72-2010
Wolcott, R. M.
72-1909
72-1999
Wolfe, H. R.
72-1675
72-1676
Wolverton, B. C.
72-1879

Wood, N. F.
72-1574
Woodside, M. D.
72-1993
Wright, R. M.
72-1817
Wright, S. J. L.
72-1752
Wurster, C. F.
72-1483
Yagi, T.
72-1384
Yamaguchi, M.
72-1472
Yamamoto, I.
72-1870
72-1947
Yamamoto, N.
72-1837
Yamamoto, O.
72-1583
Yamane, V. K.
72-1855
Yamashita, T.
72-1804
Yanagisawa, M.
72-1931
Yang, S. F.
72-1472
Yano, Y.
72-1442
Yap, H. H.
72-1488
72-1978
Yarbrough, J. D.
72-1501
Yasugi, N.
72-1683
Yokota, T.
72-1552
Yomtov, M.
72-1718
Yoneda, H.
72-1779
Yonemura, J.
72-1641
Yoneya, T.
72-1837
Yoshinaga, E.
72-1381
Yoshizaki, H.
72-1731
Young, J.
72-1436
Young, S. J. V.
72-1558
Youngs, W. D.
72-1392
Yu, C.-C.
72-1499
Yu, T. C.
72-1864
Yudina, T. V.
72-1946
Zabel, E.
72-1667
Zabik, M. J.
72-1424
72-1473
72-1871
Zaika, A. P.
72-1528
Zatz, J. L.
72-1756
Zaylskie, R. G.
72-1872
Zetterberg, G.
72-1467
Zimmerman, A.
72-1744
Zitko, V.
72-1416
72-1418
Zlateva, M.
72-1770

Center for Disease Control
72-1362
Entomological Society of Canada
72-1827
Sect. of Labor Hygiene,
Div. of Safety and Hygiene,
Japan Ministry of Labor
72-1701

Use of funds
for printing this publication
approved by
the Director of the Office of Management and Budget
March 31, 1970

ORDER FORM
FOR HAPAB

To: Superintendent of Documents
Government Printing Office
Washington, D.C. 20402

Please enter my subscription for Health Aspects of Pesticides Abstract Bulletin. I am enclosing Money Order ☐ Check ☐ for this subscription. ($6.50 a year; $1.75 additional for foreign mailing.)

Please address the HAPAB as follows: _____

_____

_____

UNITED STATES GOVERNMENT PRINTING OFFICE, WASHINGTON, D.C. 1968

For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington, D.C. 20402. Subscription price $6.50 a year, $1.75 additional for foreign mailing. Price for a single copy of this issue is 45 cents. Index issue $1.25.

ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF PESTICIDES PROGRAMS
Division of Pesticide Community Studies
4770 Buford Hwy., Bld. 29
Chamblee, Georgia 30341

POSTAGE AND FEES PAID
ENVIRONMENTAL
PROTECTION AGENCY
EPA-335

If you do not desire to continue receiving this publication, please CHECK HERE ☐; cut off this label and return it to the above address. Your name will then be removed from the mailing list.